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SURGERY OF THE PANCREAS

WITH ESPECIAL CONSIDERATION OF ACUTE PANCREATIC NECROSIS[1]

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A QUARTER of a century ago, in a lecture before the Congress of American Physicians and Surgeons on "The Present Status of Pancreatic Surgery with Especial Emphasis upon Injuries and Inflammations," the famous German surgeon von Mikulicz, designated pancreatic surgery as the least complete chapter in abdominal surgery. Even today there are numerous unsolved questions in practical as well as scientific pancreatic surgery. "Why do so many people die from this acute, dread disease?" is the question that has troubled the surgeon for the past half century. This is especially true since Balser in 1882 published a description of fat necrosis. Gussenbauer was the first to record a case of pancreatic surgery, his report being made in 1883. Since Halsted, in 1898, described an operative procedure for the cure of acute pancreatitis, and Koerte, in the same year, set forth his very comprehensive and fundamental work which seemed to cover all of the then current knowledge of pancreatic disturbances, very little of note has been published on the subject.

However, experimental research on the pathogenesis of pancreatic surgery has added a noteworthy chapter to the unfinished history. This experimental research aided by surgical autopsies has led to a complete understanding of the devastating effect of the pancreatic enzyme when placed extraneously, and to an understanding of the too early activation of the digesting enzyme in the gland itself coupled with autodigestion of the pancreas.

As a result of clinical observation and experimental research, there have been developed in the past 20 years, systematic operative principles which have brought about successful results in pancreatic surgery. There has been no sudden, marked progress which can be compared to the solution by the internist of his most serious pancreatic problem, namely, the treatment of diabetes through the use of insulin. Because of the physiological and pathological nature of the pancreas, this branch of surgery has developed slowly, and even now it does not seem likely that more rapid progress will be soon forthcoming.

As hypothetical theories are gradually replaced by established facts, the surgeon will approach the practical solution of the clinical problem, and will eventually evolve an ideal treatment and possibly adequate prophylactic measures.

Our opinion upon the present status of pancreatic surgery is based not only upon our own experiences and the reports in the literature, but upon a study of 2,137 cases which have been treated in the past 8 years. Questionnaires were sent to over a hundred clinics and hospitals and a general report on the answers

[1] Condensed report made by Prof. Schmieden before the Society of German Surgeons in 1927, with illustrations and tables from the Arch. f. klin. Chir., cxlviii. Translated by Samuel Fogelson, M.D., Chicago.

was presented by von Schmieden at the Society of German Surgeons in 1927. It is impossible to give here a complete review of all of the information obtained, so we will limit our discussion to the subject of pancreatic necrosis.

The anatomicopathological picture (Fig. 1, frontispiece) shows simultaneous necrosis, inflammation, and hæmorrhage. In a discussion as to which of these plays the primary rôle, Opies and Gulekes are of the opinion that necrosis is the dominating factor. This opinion has come to be the generally accepted one. The earliest studies naturally show the unmixed picture. But from this, one cannot conclude that the all-important "pancreatic drama" may not be secondary to a primary inflammation—pancreatitis—with necrosis and hæmorrhage secondary, or that hæmorrhage, as a result of syphilis or arteriosclerosis (pancreatic apoplexy) may be a primary pathological change. Hæmorrhage is followed by necrosis and inflammation due to invasion of the pancreatic enzyme which immediately becomes activated. The explanation of both Bergmann and Guleke is of utmost importance in understanding the pathogenesis of this process. They believe that the symptomatology and course of the disease is secondary to an intoxication from the autolytic destruction of the pancreas. The trypsin itself is not the direct causative factor, but it is a protein formed by the pancreatic autolysis. It is rapidly absorbed and death results in a few hours before resistance is developed either by an inflammatory process or by antitoxins.

The changes in the pancreas vary tremendously in type and degree. Early alterations consist of œdema, often bile stained, with a serous infiltration of the entire body of the pancreas. There may still be, however, a complete recovery without damage to the parenchyma (Zoeppfel). In an advanced stage, the gland resembles variegated marble in color, because of the diffuse swelling and hæmorrhagic infiltration (Fig. 2, frontispiece); as a result of further destruction there develop structureless necrotic tissue masses, ranging in color from brown to dull gray, so that ultimately the gland assumes a cooked appearance. In the presence of excessive hæmorrhage, there may be a complete disappearance of the tissue structure and the pancreas is changed to a blue-red blood clot. The typical disseminated spots, chalky and opaque, of fat necrosis may appear within 16 hours. These spots together with the peritoneal effusion, give the surgeon a diagnosis pointing to the pancreas as the causative organ. Langerhans, Hildebrand, and Flexner believe that the enzymic action rather than the bacterial action is the cause for the splitting of the fats, the changing of the neutral fats to sodium and calcium soaps. These are found in the gland itself, in the peripancreatic fat, and scattered throughout the entire peritoneal cavity, as well as the retroperitoneal, subpleural, mediastinal, pericardial, and even subcutaneous tissues; they have even been described symmetrically scattered about in the extremities (Jenkel).

The blood stream is the only accepted means by which the enzyme which splits the fat in the atypical, distant places is spread (Eppinger, Payr, and Martina). In the peritoneal cavity the reaction is due to direct contact with the enzyme, and most frequently the toxin is conducted by the lymphatics to other areas. Rostock, working in Guleke's clinic, has recently demonstrated this by new and important evidence.

Should this pathology be survived, there still is the further danger from the extension of the process to the peritoneum (peritonitis toxica) and further, to the retroperitoneum, and eventually, it is possible, even to the pleura (Clairmont). In these locations, the peritoneal exudate containing the enzyme, acts in its usual manner and leads to tissue destruction, a primary sterile inflammation with a secondary bacterial infection, the original bloody serous exudate becoming purulent. As a result, the glandular parenchyma, being poor in connective tissue, rapidly disintegrates, giving off pancreatic sequestra, which subsequently drain out in masses together with the operative discharge. The organ may become swollen to the size of a man's fist, and the patient is endangered by still other complications. In the prolonged, subacute type of the disease, there may be a

downward seeping of the pus, and, if the patient survives the intoxication and peritonitis, he is still liable to suffer from a pyæmia, with or without mesenteric thrombosis, or else he may suffer from an involvement of the portal veins.

The normal protective tissue reaction is everywhere decreased by the corrosive action of the gland secretion. There may occur: perforation of such viscera as the stomach or the bowel; ruptures in the neighboring body cavities; or, severe, even fatal, hæmorrhage of the numerous blood vessels surrounding the gland. Occasionally a case may develop a pancreatic pseudocystic disintegration cavity which should be recognized as part of the healing process.

In spite of the fact that the methods of pathogenic development are numerous, the etiological causes will be found to be even more frequent. Neither experimental results nor clinical observations have solved the problem of prophylaxis from an etiological aspect. We present here only those experimental results which, in dogs, either through mechanical or toxic tissue damage, or by blood-vessel and tissue changes, have produced the homologue to the human clinical picture. The initiating factor for the development of the morbid process has, however, not yet been demonstrated in a manner free from all possible criticism. We do know that the lymph channels and blood vessels are the most important paths through which the most destructive agents flow (Fig. 3).

There have been clinical observations of all these etiological possibilities. Duodenal diverticulitis is one of the least recognized factors in acute pancreatic necrosis, but its genesis has been forcibly established by the work of Forssell and Key, Case, Wilkie, Akerlund, and Clairmont.

Of greater significance are the variations in the pancreatic ducts and their mouths. Osler explained these variations in the relations by the embryological development of the gland. Clairmont differentiates ten major types. By means of iodine injections in the tail of the pancreas in cadaver preparations, we have been able to demonstrate even more anomalies of the duct system.

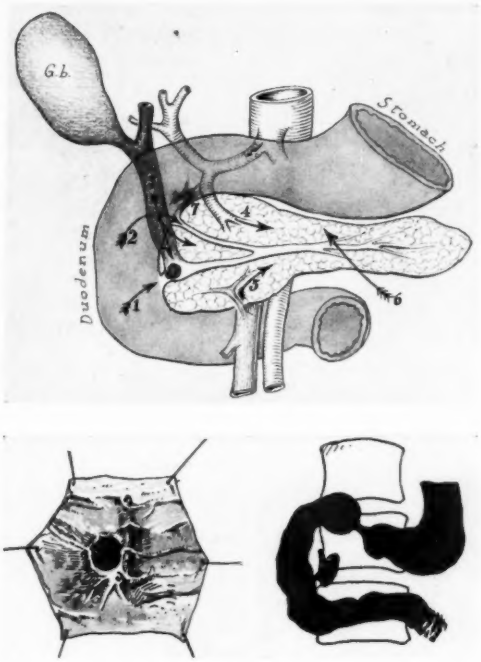

Fig. 3. Schematic description of the avenues of development of pancreatic necrosis: *1*, through the duct of Wirsung from the duodenum; *2*, through the duct of Santorini; *3*, from the biliary ducts; *4*, from the arteries; *5*, from retrograde emboli through the veins; *6*, traumatic; *7*, from duodenal ulcers through the lymphatics. Fig. 3. a (lower left). Duodenal diverticula near Vater's papilla. b (lower right). Roentgen demonstration of diverticulum by von Akerlund.

Opie was the first to find at autopsy, a small gall stone fixed in the ampulla of Vater, and it was he who explained the development of acute pancreatic necrosis as secondary to the back flow of bile into the pancreatic ducts. Since then the topographical course of the ductus choledochus and the duct of Wirsung, together with their different types of union, have been given the utmost consideration (Mayo-Robson, Osler, Bunger, etc.).

The results of research concerned with the examination of the conditions in which bile can invade the pancreatic ducts are extremely contradictory. While Mann and Giordano found it anatomically possible for a gall stone to block the papilla in such a manner that bile may invade the pancreatic ducts

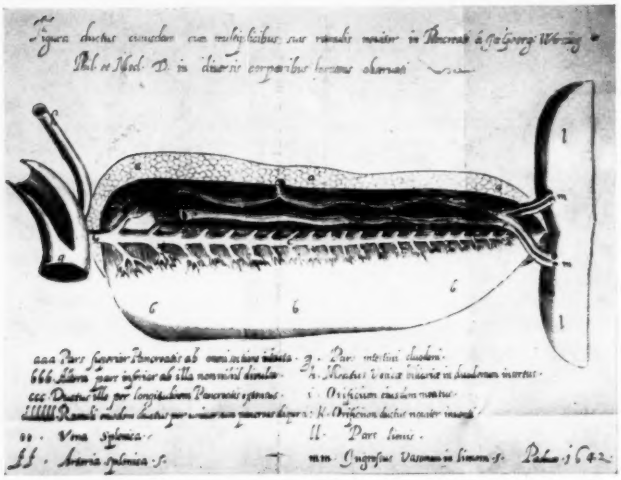

Fig. 4. Wirsung's description of the pancreatic duct discovered by him. Copy of an etching sent by Wirsung to his teacher, Riolan (1642). Reduced copy from Schirmer (1893).

in 3.5 per cent of the cases; Judd found it to be true in 4.5 per cent of the cases; and, likewise, Cameron and Nobel, in 66 cases in 100 examined. Our investigation demonstrated that in 8 of 35 cases, it was possible after clamping the papilla of Vater to fill the tail of the pancreas from the bile ducts, or vice versa.

Our clinical observations are of particular value, inasmuch as 7 cases of papillary stones were found among 31 cases of acute pancreatic necrosis with an associated cholelithiasis (Fig. 5).

The recognition of the extreme significance of disease of the biliary ducts in the etiology of acute pancreatic necrosis is not only of marked importance in understanding the pathogenesis but also for diagnosis, therapy, and prophylaxis.

While it has been customary to look upon the simultaneous presence of cholelithiasis and pancreatic necrosis as coincidental, it is noteworthy that this combination is more frequently found when the surgeon looks especially for this possibility. This is definitely demonstrated by a comparison of the figures collected from our questionnaire with those of Guleke. There were 894, 69.8 per cent, cases of biliary duct disease in 1,278 cases of pancreatic necrosis operated upon, whereas Guleke's questionnaire in 1924 comprising 437 cases had 208, or 59 per cent, of simultaneous cholelithiasis (Fig. 6). The percentage of fixed choledochus or papillary stones in the Guleke statistics was surprisingly small, while in our report there were 174 common duct stones and 57 papillary stones. Our own material, in which the rôle of the biliary passages was especially noted, shows 31 cases of biliary stone in 38 cases (81 per cent), and of these 15 were common duct stones, 7 of which were in the papilla.

In what fashion then does the diseased gall bladder injure the pancreas? The clearest cases are those in which acute pancreatic necrosis results from the fixation of a stone in the ampulla of Vater. The same mechanical damage occurs here as in the experimental production of acute pancreatic necrosis. Stasis of the secretion and elevation of the pressure in the pancreatic ducts together with chemical trauma from the bile and bacteria are the provocative factors. The passage of concretions with their temporary fixation of the ampulla of Vater may act in the same manner. At the time of operation, the stone may be found in the bowel (Jung, Zoepfel, Reich). It may not even be found at autopsy, so that definite proof of its existence may be

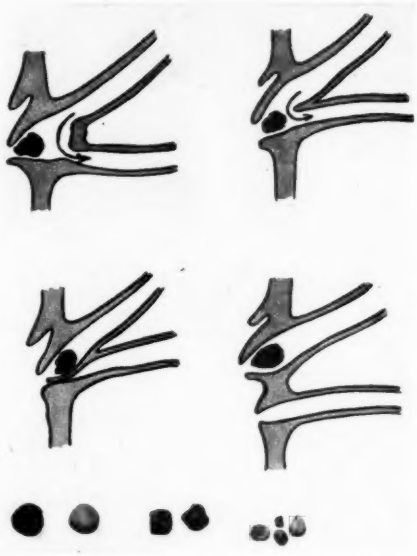

Fig. 5. Obstructive stones in the papilla of Vater and the various types of mouths of ductus choledochus and pancreatic ducts. Below are the stones which have caused the acute pancreatic necrosis contrasted with pepper berries.

lacking. On the other hand, the stone may fall back into the dilated common duct after being temporarily fixed at the ampulla. Then there is still the possibility that the successful passage of a stone through the papilla may stretch it and cause it to gape, so that the duodenal content may invade the duct of Wirsung and activate the pancreatic enzyme. Stones which are situated in the supraduodenal part of the common duct can have no direct mechanical effect upon the pancreatic ducts, but may induce spasm of the sphincter of Oddi, so that regurgitation of bile into the pancreatic ducts results. A diseased gall bladder—a cholecystitis—may produce the same effect even though the common duct is free from stones. Obstruction of the common duct above the papilla may cause bacteria and an exudate containing bile from the intrapancreatic biliary ducts to invade the adjacent pancreatic tissue and may give rise to an associated pancreatitis. Finally, invasion of the lymph glands in the head of the pancreas from the lymphatics of the gall bladder may

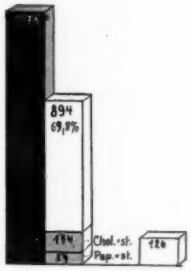

Fig. 6. The overwhelming significance of biliary-duct diseases as contrasted with all other etiological factors in the cases of acute pancreatic necrosis operated upon in our collected statistics.

lead to stasis and activation of the pancreatic enzyme.

In occasional cases it is often difficult to demonstrate the genetic connection between cholelithiasis and pancreatic necrosis; but its tremendously greater frequency, as contrasted with all other known etiological factors, justifies the assumption of pancreatic necrosis as a complication of cholelithiasis. Early operations for cholelithiasis are therefore amply justified (prophylaxis). As a rule pancreatic necrosis follows a neglected chronic cholelithiasis (important for diagnosis), but this grave disease can occur early without any evidence of a common duct complication (acute pancreatic necrosis following labor in a primipara). In our youngest patient, 13 years old, the unmistakable etiological factor, was a stone about the size of a pepper berry in the papilla.

Undoubtedly, a minute examination of the biliary ducts is a definite prerequisite in every case of pancreatic necrosis operated upon. The correct methods of treatment have been established as far as the treatment of gallstone disease demands. The purpose of this part of the operation must, therefore, be to conduct the bile away in the quickest and simplest manner and to drain the pancreatic ducts. Rapid gall-bladder drainage will suffice if the large bile ducts are free from stones. If the condition of the patient will permit, we agree with Nordmann, von Haberer, and Zopfer that the operation of choice is cholecystectomy with choledochostomy. Cholecystectomy alone is unsatisfac-

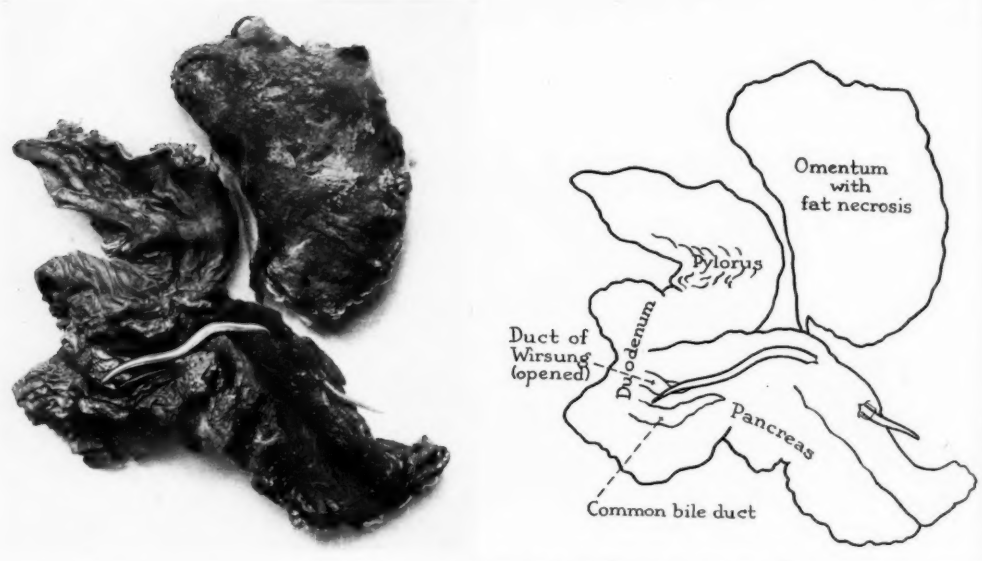

Fig. 7. Acute pancreatic necrosis caused by invasion of the duct of Wirsung by ascaris. The patient, 50 years of age, was operated upon 6½ hours after the onset of the disease. Death occurred 14 hours after operation.

tory as it not only hinders drainage but increases the pressure in the biliary duct system. Internal biliary duct anastomoses (choledochoduodenostomy) must be condemned because the peritoneum in the neighborhood of the pancreas has poor adhering qualities, and at this point all intestinal sutures are unreliable. Moreover, such operations take too much time. The statistics in our large series demonstrate the favorable results from the different biliary operations performed and invalidate the conclusions of Gegner who advises against any intervention upon the biliary passages fearing the resulting disadvantages (Table I).

TABLE I.—OPERATIONS PERFORMED IN CASES OF ACUTE PANCREATIC NECROSIS IN WHICH THE BILIARY OPERATION WAS DONE AT THE SAME TIME

	Recovered Per cent	Died Per cent
Cholecystostomy	55.6	44.4
Choledochostomy	60.0	40.0
Cholecystectomy and drainage of the common duct	64.0	36.0
With biliary care	61.3	38.7
Without biliary care	40.7	59.3
Cholecystectomy	49.4	50.6
Choledocho-enterostomy	20.0	80.0

A study of the literature shows that 50 cases of invasion of the duct of Wirsung by ascarides had results similar to those in cases with stone incarcerated in the papilla (Eberle, Pfanner, Gallie and Brown, Righy, Sabrazes, Seyfarth).

In this connection I wish to report briefly a very rare case of acute pancreatic necrosis due to invasion of the pancreatic ducts by ascarides.

The case was operated upon with a tentative diagnosis of acute pancreatic necrosis 6½ hours after the onset of excruciating pain, uncontrollable emesis, and immediate shock. The entire pancreas was a blue-red color, resembling a hæmorrhagic infarct, but fat necrosis had not yet occurred. The

TABLE II.—PANCREATIC DISEASE AFTER OPERATION ON THE BILIARY SYSTEM

	Cases
After cholecystectomy	24
After supraduodenal choledochotomy	9
After transduodenal choledochotomy	3
After choledocho-entero-anastomosis	2
Total	38

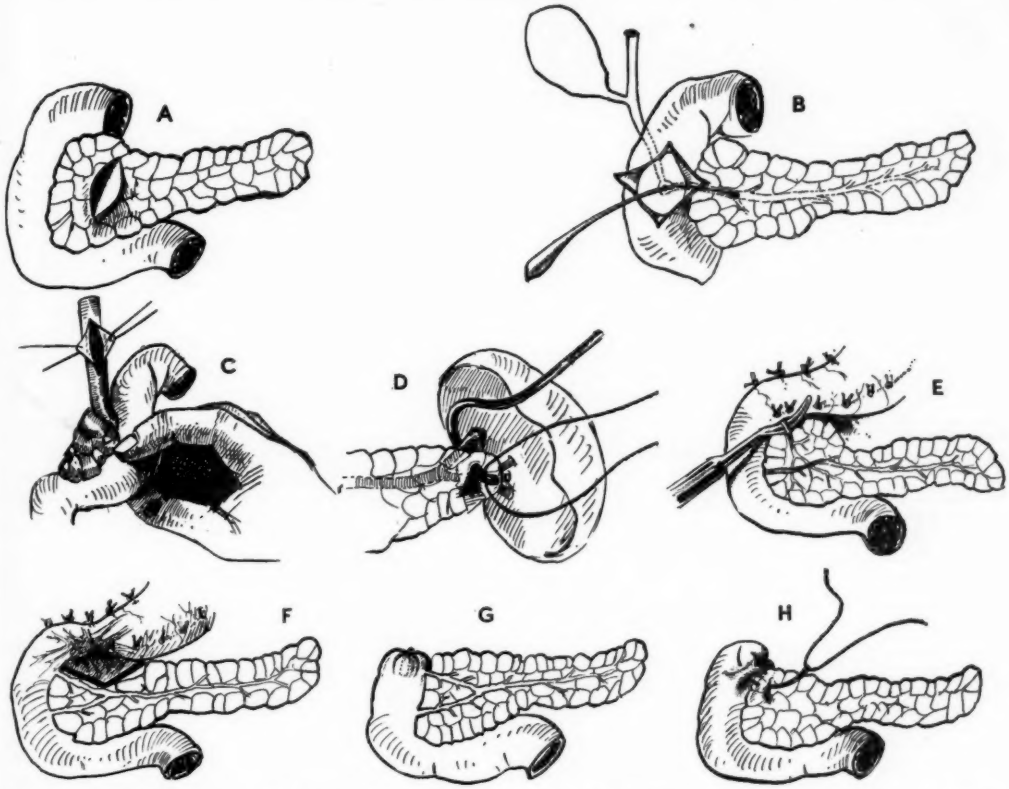

Fig. 8. Operative measures which can lead to acute pancreatic necrosis: A. Diagnostic excision from pancreatic substance. B. Slipping of sound toward the left and into the pancreatic duct in transduodenal choledochotomy. C. The Kirschkern maneuver for pushing up gravel or stones, with injury to the tail of the pancreas during ligation of the splenic vessels. E. Tearing of the duct of Santorini during separation of the duodenum. F. Sharp excision of a penetrating ulcer from the pancreatic substance. G. Deep inversion of the duodenal stump. H. Covering of the duodenal stump with the pancreatic head. The sutures are too deep and therefore invade the pancreatic substance.

typical hæmorrhagic exudate was present in the free peritoneal cavity, but there were no stones in the biliary ducts. Death occurred 14 hours after the operation. The autopsy was done immediately and revealed extensive fat necrosis in the omentum and mesentery, which had not been seen during the operation; hæmorrhagic infiltration of the entire pancreas with tiny microscopic areas of necrosis; a single ascaris was found in the duct of Wirsung, its head reaching to the tail of the pancreas and its tail protruding 1 centimeter into the lumen of the common duct. It was still alive and mobile when the ducts were opened. There were no other worms in the entire gastro-intestinal tract.

Next to stasis of secretion and its activation, the tissue damage, secondary to the violent penetration by the worm, was an important factor in the production of acute pancreatic necrosis. This case is an example of its traumatic genesis and the extremely varied forms of postoperative pancreatic necrosis.

There is no doubt today that an operative trauma which injures tissues should receive great attention. We will later point out a large number of measures, such as *vitia artis*, which have been considered harmless but which we would advise the careful surgeon to avoid. The pancreas should always be considered a labile organ which can be readily injured at operation.

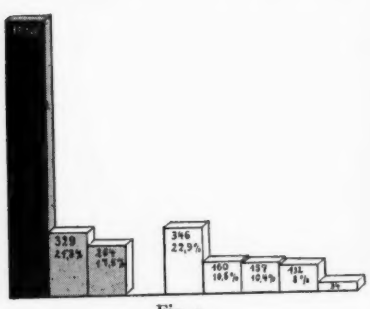

Fig. 9.

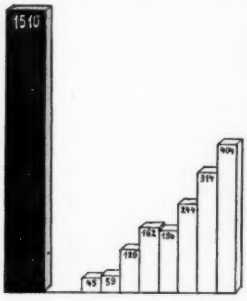

Fig. 10.

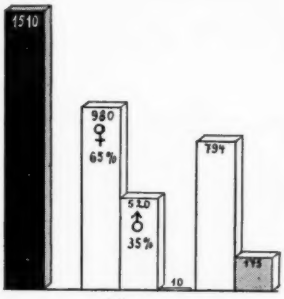

Fig 11.

Fig. 9. The pre-operative diagnosis and the errors in diagnosis in our compiled statistics.

Fig. 10. Collected statistics from one hundred four clinics and hospitals showing the total number of cases of acute pancreatic necrosis which have been studied during the last 8 years.

Fig. 11. Showing the marked preponderance of females over males, and obese over thin patients.

The futility of many half-hearted operative procedures was demonstrated in a number of discouraging cases. Even the blunt trauma which follows retroduodenal palpation of the common duct or incision for stones, which may result in the pushing up of the stones and the so-called "Kirschkernmanoever," which is so generally used, may lead to fatal complication. Pancreatic necrosis may follow the closure of the suture-knot after a bougie has been passed through the ampulla of Vater (Rost). At one of the last surgical congresses, Walzel demonstrated the danger which may follow the use of a sound in transduodenal choledochotomy, if the pancreatic ducts are accidentally invaded. He warns especially against this immediately before the transduodenal removal of a stone in the ampulla (Table II).

TABLE III.—PANCREATIC DISEASE FOLLOWING OPERATION

	Cases
After stomach operation	91
After biliary operation	38
After splenectomy	7
After diagnostic excision of the pancreas	4
After other operations	5
Total	145
Type of pancreatic disease	
Acute pancreatic necrosis	58
Purulent pancreatitis	15
Pancreatic abscess	10
Pancreatic fistula	18
Fat necrosis without any evident pancreatic change	3
Total	104

An acute pancreatic necrosis may follow the blunt trauma occurring during gall-bladder operation and also the intentional or accidental damage to the gland or neighboring organs during operation; after diagnostic biopsy; after suture of the pancreas to the duodenal stump in a Billroth II operation; after puncture; secondary to excision of the base of ulcers (Clairmont); after the slightest damage to the tail of the pancreas; secondary to splenectomy (Ranzi, Walzel, Koerte) or during the ligation of the right kidney pedicle (Young) (Fig. 8).

These graver complications do not always occur, and as a general rule only those are recognized which terminate fatally. Instead of acute pancreatitis, a purulent pancreatitis, abscess formation, or a pancreatic fistula with only a temporary disturbance of function, may result (Table III). Clairmont has demonstrated lung complications in connection

TABLE IV.—STOMACH OPERATIONS LEADING TO PANCREATIC DISEASE

	Cases
Excision of penetrating ulcer	20
Mobilization of the duodenum	17
Inversion of the duodenal stump after Billroth II	16
Ligation of the duct of Santorini	11
Resection of pancreatic tissue invaded by stomach carcinoma	11
Suture ligature of bleeding pancreatic vessels	8
Covering of the duodenal stump by head of pancreas	6
Gastro-enterostomy	2
Total	91

with pancreatic trauma. Fifteen per cent of all cases of duodenal resection develop serous pleurisy which, however, disappears in 3 to 4 days. It is unfair to assume that all these operations were poorly done. Many surgeons adopt every precaution, but one generally accepted warning must be mentioned here, namely, a reference to a generally rare, but grave, danger.

When the pancreas has been involved in the operation, it is certainly not advisable to close the peritoneal cavity without drainage. This measure affords at least some measure of protection.

Stomach and duodenal operations supply quite a different group of pancreatic complications (Clairmont, Table IV): peritonitis may follow suture-line leakage, which is secondary to the digestion of the technically perfect sutures by the pancreatic enzyme which arises from an unrecognized injury to the pancreatic veins. The mobilization of the duodenum supplies the source of blunt trauma which injures the parenchyma of the pancreas. Excision of the ulcer and faulty handling of the duodenal stump have already been mentioned.

Partial resection of the gland may be done for advanced cancer of the pancreas. This was recommended by Finsterer contrary to the existent knowledge of the high incidence of pancreatic necrosis and peritonitis. Von Mikulicz figured that this operation elevated the mortality from 27 to 70 per cent, but today the danger does not appear so great. Because of the same high mortality associated with pancreatic resection for other causes, progress seems to be at a halt in this field of surgery.

Anomalies of the ducts supply a separate chapter. It is frequently possible without any danger to mobilize the duodenum and separate it from the pancreas as far as the ampulla of Vater. This procedure becomes hazardous when there is a possibility of impinging upon the duct of Santorini, and its ligation is of importance. This was demonstrated by the work which Enderlen suggested to von Schwarz. This research demonstrated upon an embryological basis two forms of pancreas, namely, a tongue and a hammer type; the latter usually associated with a large and actively functioning duct of Santorini.

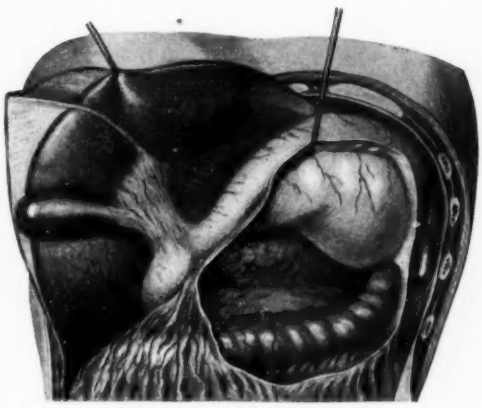

Fig. 12. Transperitoneal exposure of the pancreas through cutting of the gastrocolic ligament.

In spite of this, the duct of Wirsung may be of importance and there is a definite danger of its being ligated in cases requiring extensive separation. The following conclusions are justifiable from Schwarz's research: There is great danger of ligating the duct and obstructing its drainage in one-fourth of all cases in which the pars duodeni is freed; a large duct of Santorini draining an important part of the gland should be feared when the pancreas is firmly fixed to the duodenum or the head of the pancreas is very large (occasionally reaching to the pylorus); the tongue-shaped type of pancreas is generally associated with less danger.

These possibilities should be considered and avoided in every pyloric resection which approaches the pancreas. Regurgitation into the duodenal loop should also be avoided because of the danger of associated regurgitation into the pancreatic ducts. In addition, care should be taken to keep the duodenal stump which has been inverted into the retroperitoneal space after a Billroth II, from pressing too firmly upon the unprotected pancreas.

This chapter still contains many important unanswered questions for the practical surgeon. There is a possibility that the real cause of danger is not yet understood despite consideration of the separate rôle played by

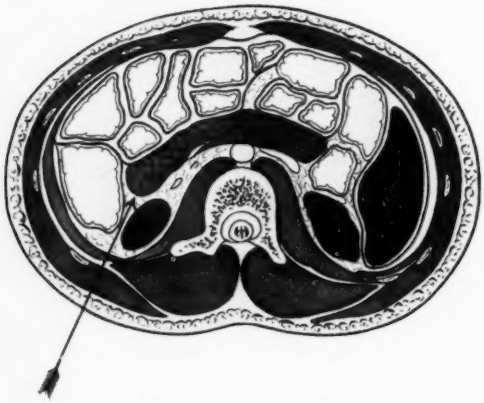

Fig. 13. Left-sided lumbar approach to the pancreatic tail.

each type of anatomical anomaly, together with the elimination of danger involved in surgical procedures. We may call this a predisposition to pancreatitis, a condition readily understood when we consider that in 1 case a biopsy will lead to fatal complications while in 99 other cases, there will be an uneventful convalescence.

Gulek's experiments have demonstrated that there is a marked significance in the phase of secretion, but that there is still a predisposition to pancreatic involvement; or perhaps a constitutional predisposition such as exists in the alcoholic, adipose, or luetic. Perhaps we are right when we consider the sudden onset of this condition as the cumulative effect of all the etiological danger factors. It must be our goal to give this line of etiological reasoning our intensive study.

A consideration of those etiological factors which may damage the pancreas because of an imperfect blood supply indicates that they may include nutrition of the gland due to impaired circulation secondary to spasm, thrombosis, embolism, arteriosclerosis, or to lues. Postoperative pancreatic necrosis, secondary to retrograde emboli, belongs in this category (Jenckel, Orthner); and may follow any operation in the peritoneal cavity, occurring either without manipulation of the pancreas (resection of bowel, omentum, or ovary) or when the operative field is distant from the pancreas (prostatectomy, or operation for varicose veins). It may follow intoxication through the blood stream either in cases of severe poisoning from lysol, oxalic acid, meat, or sausage poison, or, as frequently happens, after septic processes such as puerperal sepsis, typhus, measles, osteomyelitis, or parotitis. Here again we are impressed with the fact that only certain individuals of an entire group who are affected with the same disease actually develop this complication. We know no causative factors for its development and moreover we do not know why after the introduction of the same septic process in several individuals, one patient will develop an acute pancreatic necrosis with its stormy course and another will develop a purulent pancreatitis which behaves as might a pyæmic abscess were it located elsewhere. The resistance of the individual and development of immunity, together with the local and bacterial conditions naturally have a definite consideration in the prognosis (Zoepfel, Brutt).

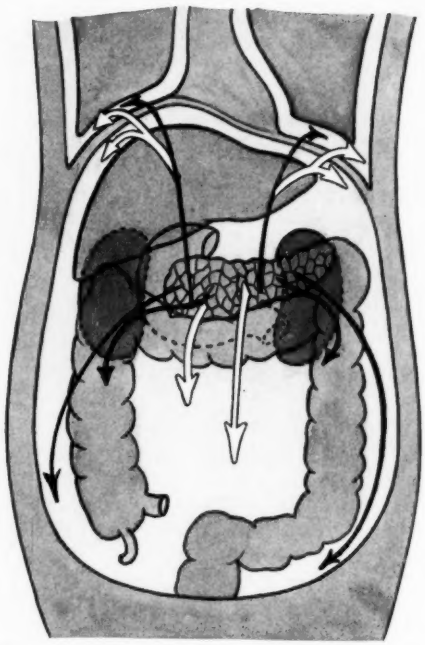

Fig. 14. Typical spread of pus from the pancreas. The red arrows indicate the intraperitoneal course. The black arrows indicate the retroperitoneal pathways.

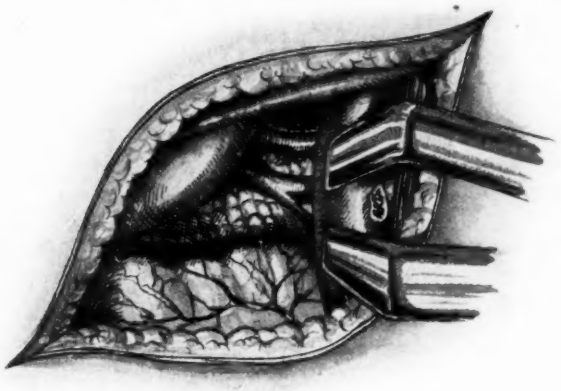

Fig. 15. Exposure of the pancreas through a left lumbar incision and median traction of the kidney. The spleen is visible on the left border of the incision. The splenic artery and vein cross the pancreas which is posterior.

The chapter on bacteriology is purposely slighted because at present it is not possible to evaluate the significance of infection in the development of acute pancreatic necrosis. The material here analyzed was compiled from a questionnaire and did not consist of consecutive cases, so that no basis for positive conclusions was afforded (Table V).

Our own findings are based upon 10 cases: Bile from the gall bladder and common duct and the peritoneal exudate was found sterile in 9 cases. Streptococci were found in the bile and the peritoneal cavity in 1 case but may have been due to a secondary infection, inasmuch as they were found 10 days after the onset of the disease.

We will only mention the frequently discussed symptoms and satisfy ourselves with a classified grouping of the clinical manifestations. Pain of the most excruciating type is nearly always present and is an important symptom. After very mild colic-like prodromal pains (warnings which may be absent), an apoplectiform attack of pain and collapse suddenly develop. These may be the result of intoxication from the pancreatic enzyme which lies free in the peritoneal cavity; the distention of the gland capsule; infiltration of the mesocolon; or pressure upon the cœliac plexus or upon the semilunar ganglion (cœliac neuralgia). The persistence of the pain until shock develops, differentiates it from biliary colic. The pain diffuses backward from the left abdominal region to the lumbar region, radiating to the left shoulder. The pulse is at first slow and small, and later as a result of vagus irritation becomes still slower. Abdominal rigidity and distention are early signs, but there is, even at this time, little distention and rigidity as contrasted to that occurring in ileus or peritonitis. The temperature is normal or subnormal; there is agonizing belching and vomiting; while occasionally the pancreas can be palpated as a transverse ridge (Koerte).

Recognition of the disease at this stage requires a previous suspicion of the diagnosis. The observant general practitioner will recognize cases of "peritonitis without temperature or abdominal rigidity." The following symptoms are certainly suggestive: cyanosis of the face, hands, and extremities; or, as described by Walzel, blotches and lattice-like streaks of dark cyanotic color; upper abdominal distention; tachycardia, 120 to 140; slight icterus; persistent emesis; absence of bowel movements or flatus; glycosuria; and fatty stool. Melæna and emesis are rare symptoms as are oliguria and anuria which indicate kidney damage. Systemic intoxication causing

TABLE V.—BACTERIOLOGICAL FINDINGS IN ACUTE PANCREATIC NECROSIS; RESULTS IN TWO HUNDRED AND EIGHTY-TWO EXAMINATIONS

	Cases		Cases
Peritoneal exudate		Bile	
Sterile	84	Sterile	40
Bacteria	103	Bacteria	54
Bacillus coli	24	Bacillus coli	26
Staphylococci	34	Staphylococci	10
Streptococci	37	Streptococci and mixed infection	10
Frankel gas bacillus	2		
Paratyphoid bacillus and mixed infection	1		

cramps and psychic disturbances have been observed. Death results from cardiac failure and the symptom complex of paralytic ileus clouds all earlier abdominal symptoms.

In contrast to the rarer general picture, there is still lacking a modern aid for making the exact diagnosis in the numerous atypical cases. Even today we must manage without a pathognomonic test which would make the diagnosis positive and which Koerte longed for in 1911. Wohlgemuth's diastase test, in which the ferment is easily demonstrated in the urine and blood, is of some value. It should be critically considered even when carefully done for even then the failures run about 20 per cent (Table VI).

In most instances, one should rely upon the most probable diagnosis, and differentiate between gall-bladder perforation, stomach and duodenal perforation, gangrenous appendicitis, peritonitis of unknown etiology, ileus, mesenteric thrombosis, and ureteral colic. Our compiled statistics show that a positive diagnosis of acute pancreatic necrosis was made in only 21.8 per cent (Fig. 9) of the cases and suspected in 17.5 per cent. Therefore, the pancreas was considered only in little less than 40 per cent of the cases.

The most common source of error in our series was acute cholecystitis which occurred in 22.95 per cent. This is readily understood as the patient early in the disease creates the impression of this diagnosis by giving a history of repeated similar transient attacks from which he completely recovered. On the other hand this is a fatal diagnosis, for the physician becomes satisfied with the diagnosis

TABLE VI.—DIASTASE RESULTS IN THE URINE IN NINETY-SEVEN CASES OF ACUTE PANCREATIC NECROSIS AS DETERMINED BY TWENTY-ONE DIFFERENT EXAMINERS*

	Cases
Increased excretions	78
Normal excretions	19
(20 per cent error)	

*Normal: 1 c.cm. of urine d'gests 64 c.cm. of 1% diastase solution in 30 minutes at 38 degrees C.

of gall stones, orders morphine, and resorts to surgical intervention only when the pain is found to persist and the patient's condition is much worse. The influence of the biliary history together with the existing icterus and absence of temperature, causes further delay in those cases in which there are no marked signs of peritoneal irritation. When the condition becomes advanced, a pre-operative diagnosis of phlegmonous cholecystitis or perforation of the gall bladder, is first made and then the patient is prepared for operation.

Even today, we do not give enough attention to Koerte's old warning to consider acute pancreatic necrosis in all cases developing acute abdominal diseases especially when biliary colic has been evidenced. The recognition of the close genetic connection between cholelithiasis and pancreatic necrosis should cause the general practitioner to suspect implication of the pancreas especially in the presence of an unusually severe, protracted colic, in which the pain radiates toward the left and one dose of morphine does not give relief. A gall-bladder history should afford the possibility of an early diagnosis.

Today, the surgeon sees acute pancreatic necrosis quite frequently. Thirty-eight cases were operated upon in the last 8 years, and in the same time there were 63 cases of gastric perforation—nearly twice as many! In 1903, Mikulicz reported 75 cases collected from the literature; in 1911, Koerte collected 103 cases; in 1924, Guleke reported a compilation of 437 cases; our questionnaire which was limited to post-war cases afforded 1510 cases. These statistics, together with the simultaneous solution of many individual problems, afford a reliable perspective of the position which is held at the present time by pancreatic surgery.

TABLE VII.—THE OPERATIVELY DETERMINED DEGREE OF PANCREATIC PATHOLOGY AND THE CURES FOR THE VARIOUS STAGES[1]

		Cured Per cent	Died Per cent
9.2	Œdema without fat necrosis	76.3	23.7
35.4	Œdema with fat necrosis	62.5	60.1
25.1	Hæmorrhagic infarcts	36.1	56.9
19.3	Necrosis and areas of softening	32.0	68.0
3.4	Sequestration	43.1	63.9
7.6	Abscess formation	39.9	37.5
	Averages	48.8	51.2

[1] The figures in the first column give the percentage of cases of the respective stage which came to operation.

There is a considerable increase in the number of cases occurring each year. This is especially noteworthy when compared with the well known observation by Wilms of a decrease during the years of the war. We do not believe that this increased frequency is secondary either to overeating, alcoholism, or other predisposing etiological factors but we believe that it is due to the modern viewpoint of operating upon all acute abdominal conditions as early as possible. Our statistics show a marked preponderance of 797 plethoric individuals in contrast with 175 of an asthenic type.

The heretofore generally accepted opinion that males are commonly affected is in no way substantiated by our statistics (Fig. 11), which show 980 women and 520 men. This preponderance is readily explained by the greater incidence of cholelithiasis in women.

That this disease may occur at any age was demonstrated by the fact that in our series there were 10 children. Of significance was the coincidential presence of biliary disease in 4 of the 10 cases. Stones were found in 3 young girls, 10, 13, and 15 years old, and there was a bacillus coli infection in a 6-year-old girl. Unfortunately, the other cases gave no etiological factor.

Of the 1,510 cases, 1,278 were operated upon. There were 232 cases not operated upon: in 149 cases the patients were already moribund, and in 83 cases, recovery was spontaneous because of the mildness of the condition. Only those cases in which operation definitely established the diagnosis should be discussed here. Because of the fundamental nature and course of the disease, the diagnosis must always remain doubtful in the cases not operated upon.

TABLE VIII.—COMPLICATIONS FOLLOWING ACUTE PANCREATIC NECROSIS

	Cases
Diabetes (3 deaths in coma)	18
Chronic pancreatitis	6
Recurrences (6 occurring within 2 months after operation)	26
With associated treatment of the biliary ducts	7
With treatment of pancreas only	19
Re-operation (10 cured; 7 died)	17
Cyst formation	4
Persistent fistula	5
Adhesions	3

On the other hand, there is no doubt that occasional cases of acute pancreatic necrosis may heal spontaneously when the process is favorable and the changes limited. Such cases of spontaneous healing are repeatedly reported in the literature. We ourselves have unexpectedly found residual evidence of pancreatic necrosis in 3 cases operated upon for cholelithiasis. These findings consisted of encapsulated areas of non-irritating fat necrosis located in the mesentery and omentum, and on the pancreas. Despite the fact that the acute stages of the intoxication may be survived by conservative treatment, there occur such complications as areas of softening, pseudocyst formation, or burrowing of pus which generally necessitate surgical intervention at a latter period.

INDICATIONS AND BEST TIME FOR OPERATION

The question of the indications and best time for operation is comparatively easy to answer, inasmuch as every classic case is operated upon as soon as possible. However, it is possible neither to predetermine nor to establish therapeutically which case will be severe. The advice derived from experience is to operate upon all doubtful cases. The fact that a patient may have successfully recovered from numerous similar prodromal attacks is not a contra-indication to operation. Our experience has shown that cases without premonitory symptoms are the most grave. The firmly established fact that 95 per cent of the cases not operated upon die, is sufficient indication of the gravity of the prognosis. Furthermore, it is not possible to judge the

TABLE IX.—CHRONIC PANCREATITIS IN THREE HUNDRED AND EIGHT CASES

Symptoms	Cases
Colic-like pain	173
Jaundice	127
Disturbed secretion	26
Glycosuria	23
Duodenal stenosis	21
Primary disease	
Biliary-duct disease	181
Duodenal ulcer	20
Gastritis	4
Acute pancreatic necrosis	3
Surgical procedure	
Biliary-duct drainage	129
Biliary-duct anastomosis	50
Splitting of the pancreatic capsule	106
Splitting of the stroma	27
Stomach operation	5
Exploratory laparotomy	7
Results	
Cured	239
Improved	33
Still ill	17
Died	19
Complications	
Deaths from acute pancreatic necrosis	3
Pancreatic fistula	2
Pancreatic cyst	1

TABLE X.—PANCREATIC CYSTS

Etiology	Cases		Cases
Trauma	16	Anastomosis with gall bladder	1
Acute pancreatic disease	28	Anastomosis with jejunal loop	2
Not known	73	Puncture and roentgen therapy	1
Tumor	11	**Results**	
Operative procedure		Cured	112
Obliteration by sutures	95	Died	15
Extirpation	21	Improved	1
Splitting and tamponing	5	Recurrence	3
Partial extirpation	3	Diabetes	3

severity of the disease from the clinical picture before operating.

It is likewise impossible to say how the cases which are treated expectantly will terminate. It is possible that a case which is very mild in the beginning may later become very severe. Thus, the most opportune time for operative cure has been lost. It is needless to say that inasmuch as the course of the disease is indeterminate, delay always entails great risk.

We believe that even shock should not be considered a contra-indication. Koerte and Guleke have already insisted upon the wisdom of early operation from conclusions afforded by their experience and statistical material. We wish again to emphasize strongly the urgency for early operative intervention.

We have previously demonstrated that the operative results depend upon the amount of pancreatic involvement. Unquestionably the best prognosis is to be expected in those cases in which the pancreatic necrosis is still in the early œdematous stage. With necrosis and regional liquefaction, the mortality reaches its crest only to decline again with the stage of sequestration and abscess formation. Our statistics, however, still further demonstrate that unfortunately up to now, only a very small percentage of all cases come to operation in the stage favorable for recovery (Table VII).

The most opportune time to operate is when the disease is just beginning. Operative interference is always indicated in those cases in which abdominal symptoms develop suddenly and lead to collapse. Let us, however, consider how many patients must be operated upon without an exact diagnosis and how, year in and year out, many people must die while the timid doctor temporizes believing he must wait for a classic clinical picture.

In what manner should we handle the suspected or definitely diagnosed acute pancreatic necrosis? A few preparations should be made to combat the collapse, and, under a general anæsthetic, the abdomen should be opened through a large median epigastric incision. All other preparations are made to facilitate rapid termination of the operation.

The goal of ideal surgery in a case of acute pancreatic necrosis is:

1. To demonstrate the pancreatic changes by freeing the pancreas until it is visible;
2. To protect against spread and absorption of the toxic tissue exudate (tissue fluid);
3. To obtain limitation of the process by freeing and draining away the exudate;
4. To provide drainage to the exterior of further profuse secretion and prevent retention and downward infiltration;
5. To remove the primary cause.

The following intraperitoneal methods of approach to the pancreas are at our disposal: (1) Through the gastrocolic ligament, between the stomach and the transverse colon and then through (Fig. 12) the lesser omental cavity—the best method; (2) through the hepatoduodenal ligament, between the liver and stomach—in moderate intestinal ptosis; (3) through the transverse mesocolon after separation of the mesenteric leaves—danger of damaging the arteria colica media; (4) through the retro-omentum by separating the omen-

TABLE XI.—PANCREATIC STONES IN TWENTY CASES

Symptoms	Cases		Cases
Clinical		Operative removal (all cured)	11
Jaundice	3	Found at autopsy	8
Colic	3	Changes of the pancreas	
Ulcer	6	Cystic	4
Diabetes	2	Sclerotic	9
X-ray		Acute necrotic	3
Stone shadow	2	Carcinomatous	1
Extragastric tumor	1	Fatty	1
Stomach cancer	1		

tum from the transverse colon (Stein); and (5) by Kocher's method of mobilizing the duodenum for unusual exposure of the head of the pancreas.

Koerte was also familiar with the left-sided approach from the lumbar region (Fig. 13) to the tail of the pancreas, and the right-sided lumbar approach to the head of the pancreas, and finally the transpleural way through the dome of the diaphragm.

It is generally the rule that only the most experienced can give a prognosis even from the condition of the exposed organ. Cases having a limited local reaction are the most favorable. Exposing the gland is insufficient; surgical interference must be radical.

The peritoneal covering and the pancreatic capsule always should be split as far as possible. In this way the retropancreatic space is made accessible to drainage. The swollen organ is palpated for induration, areas of necrosis, and, finally, for stones. It is advisable at this point to incise the gland substance, avoiding injury to the ducts. Large areas of liquefaction necrosis are best opened bluntly by the finger. The majority of authors do not believe that axiomatic splitting of the capsule is advisable in every case. Tamponing will frequently stop the bleeding from blood-vessel erosions; but this complication makes the prognosis less favorable. Hæmostatic suture ligatures are in general to be avoided because here one operates in chemically eroded tissue.

The greatest care should be used in walling off the pancreas from the neighboring structures and in establishing drainage. The tampon and drains must be placed so that they do not favor retention of secondary secretion and can be left in from 6 to 8 days.

TABLE XII.—PANCREATIC INJURIES. TOTAL OF SIXTY-TWO CASES—THIRTY CURED, THIRTY-TWO DIED

	Cases
Blunt trauma	31
Gunshot injuries	26
Stab wounds	5
Injuries to the pancreas alone (14 cured, 6 died)	20
Transverse rupture (10 cured, 4 died; 3 died within 24 hrs. after injury; 1 was operated upon and died 16 days after operation)	14
Gunshot injuries (2 cured, 1 died)	3
Pressure injuries (2 cured)	2
Fragmentation (1 died)	1
Complicated pancreatic injuries (16 cured, 26 died)	42
Organs injured at the same time	
Stomach	19
Liver	13
Spleen	7
Bowel	8
Kidney	2
Aorta	1
Vena cava	1
Gall bladder	1
Common duct	1
Diaphragm	1
Lung	1

The regional fat necrosis is undisturbed. The peritoneal exudate which may be either serous or purulent should be handled as in any other case of peritonitis. It should be removed from every part of the peritoneal cavity either by sponging or by irrigation, thus eliminating one source of toxic absorption.

Finally, it is necessary to guard against the characteristic downward seepage of pus which is associated with the pancreatic pathology (Fig. 14). Occasionally in the primary operation, it is necessary to provide counter-drainage in the lumbar region. From our own experience we know that such drainage is easily instituted in the first operation. It often becomes very difficult in the later stages of postoperative handling because of the extensive adhesions; one becomes uncertain and fears meeting the retroperitoneal and other vessels. This becomes self-evident when the anatomy involved in left-sided exposure is considered (Fig. 15). When there is a well walled-off abscess in the advanced stages of this disease, it is possible to consider only the lumbar method as primary.

A decision as to the operation of choice is most difficult to make, and it is of vital importance that the entire surgical procedure be suitable to the patient's resistance.

We have already mentioned the proper procedure when there is associated biliary-duct pathology. The biliary operation is done last, just before the abdomen is closed, and here again emphasis is placed upon a minute examination of the biliary ducts in every case. The reverse also holds true and in every operation for cholelithiasis, the pancreas also should be minutely examined.

The cases of acute cholecystitis which we have operated upon usually have circumscribed peripancreatic œdema limited to the head of the pancreas. This must be carefully looked for because it represents the first stage of an invasive process from the gall bladder, as there is as yet no fat necrosis or serous exudate. The indicated therapy upon the biliary system causes the disappearance of these findings together with those described and brought to our attention by Zopfel. Ample drainage and tamponage in the threatening area should be supplied. In this connection we are reminded of a case in which typical fat necrosis was found without any pancreatic changes. This, without doubt, was a case of spontaneous healing of pancreatic necrosis which had occurred in the previous few months.

However, even though the peritoneal cavity be opened, it is possible to overlook a case of acute pancreatic necrosis, especially in exploratory laparotomies for doubtful acute abdominal diseases. In such cases it is always necessary to think of the pancreas and to expose it as the fat necrosis may still be absent and the typical exudate be limited to the lesser omental sac. Another source of error in diagnosis which may cause the overlooking of acute pancreatic necrosis may be in assuming the incarceration of an associated hernia. The pain and the increased bulging of the hernia, secondary to elevated intra-abdominal pressure, suggest incarceration. However, the hæmorrhagic exudate is believed to be hernial fluid, the distended loops of bowel supporting the false diagnosis and primarily hiding the pathology.

What do we achieve by the surgical handling of acute pancreatic necrosis? Guleke's questionnaire in 1924 indicated a mortality of 52.2 per cent which is only slightly better than the 60 per cent mortality reported by Koerte in 1911. We have reviewed 1,278 cases with a mortality of 654 or 51.2 per cent. This result is amazing! We had hoped that the mean result would be much better in a large, new series of cases studied at a time when surgical intervention would be more efficient and when particular stress laid was being placed upon early operation. The figures of Table VII explain this very limited improvement.

These figures definitely demonstrate that an actual improvement can be achieved only when operation is performed in the stage before necrosis is present in a fully developed form. In this light we appreciate the idea of early operation and achieve its execution through organization of diagnosis. It is of prime importance that the general practitioner co-operate and at the proper time refer to the surgeon those cases in which there is the slightest suspicion of a beginning pancreatic necrosis.

The hope of receiving some aid from an immunizing agent has unfortunately not materialized. The well known experimental work of von Bergmann and Guleke, and that more recently by the Japanese, Ohno, has been of no help (Harms).

Also, there is slight prospect of securing an effective prophylactic immunizing agent in those cases in which there is great danger that the disease will develop.

As yet, we are almost helpless in those cases which have already developed a systemic intoxication. In our clinic for many years we have used intravenous injection of glucose with insulin. We believe that it may favorably combat the intoxication and recommend its use not only pre-operatively but also post-operatively for the first few days.

There are many serious dangers which still threaten the lives of the patients who survive the operation. Postoperative hæmorrhage has already been mentioned. These are rarely stopped successfully by tamponade and usually result fatally; late abscess formation and downward seepage of pus together with their course and treatment have already been mentioned. Finally there may occur empyema or numerous septic complications such as parotitis, pylephlebitis, or liver abscess.

The incision should be kept wide open for many weeks because of the persistent wound and pancreatic enzyme secretion and also because of the extremely long time required for the discharge of the gland sequestra. A temporary pancreatic fistula will naturally result, with marked digestion of the tissue around the wound. It usually heals spontaneously after a varying interval of sometimes weeks or even months.

After the surgical wound treatment comes the selection of a diet which is low in fats and carbohydrates. In this manner, due consideration is given to the impaired function of the pancreas which is spared every possible overtaxation.

What later course have those patients who pass through this severe disease? As yet we have no systematic postoperative histories from a large number of cases covering a protracted period. The late results of only occasional cases are available. From the results of the material operated upon in the Frankfort Clinic and from the literature, we may conclude that the majority of cases which are successfully operated upon remain permanently cured. The health and life of a few patients can of course be endangered by the sequelæ which may follow an acute pancreatic necrosis. These may be diabetes, chronic pancreatitis, recurrence, cyst formation, pancreatic fistula, and adhesions (Table VIII). As yet we are merely listing the results and the essential features of the surgical interference derived from our questionnaire in chronic pancreatitis (Table IX), pancreatic cysts (Table X), pancreatic stones (Table XI), injuries (Table XII).

CONCLUSIONS

1. Acute pancreatic necrosis is a disease of high mortality, with varied pathogenesis and etiology. In the etiology, diseases of the biliary tract are of the greatest significance. The genetic connection between bile-duct diseases and acute pancreatic necrosis affords special consideration regarding diagnosis, therapy, and prophylaxis.

2. The pancreas should be considered as an organ extremely susceptible to operative trauma and should be guarded against any possible damage especially during the course of operations upon the gall bladder, stomach, and spleen.

3. A present day mortality of 51.2 per cent was found in our compiled statistics in 1,278 cases during the last 8 years.

4. Only in early operations is it possible to achieve the best results in acute pancreatic necrosis.

5. Systematic postoperative examinations of the patients who have been operated upon for acute pancreatic necrosis are necessary because of the large number of complications which may develop.

6. The results derived from the surgical treatment of chronic pancreatitis, pancreatic stones, pancreatic cysts, and pancreatic injuries justify the hope for further progress in pancreatic surgery.

UTEROSALPINGOGRAPHY

Further Studies on the Action of the "Tubal Sphincter," a New Syringe for Transuterine Injections, and a Report of the Therapeutic Use of Iodized Oils within the Uterus and Fallopian Tubes

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In a previous communication published in a recent issue of Surgery, Gynecology and Obstetrics (18), I reported a survey of the literature on the use of iodized oils for diagnostic purposes, with particular reference to uterosalpingography, and also some original observations of my own. This work, I believe, establishes the value of the roentgenological visualization of the uterus and fallopian tubes after transuterine injection of iodized oils as a means of exact gynecological diagnosis in selected cases.

It is my belief that uterosalpingography not only supplements the insufflation of gas but frequently supplants it. In cases of sterility, it not only informs us whether the tubes are patent or not but also localizes the site of occlusion. Properly performed, the test outlines the uterus and various portions of the fallopian tubes with great distinctness.

For a description of the technique of the method, the reader is referred to my previous paper. The application of the test is quite simple, but strict aseptic precautions must be taken. In my experience, as in that of the great majority of other workers, the transuterine injection of iodized oil has been entirely harmless, and I have observed no manifestations of iodism or other injurious reactions from its use.

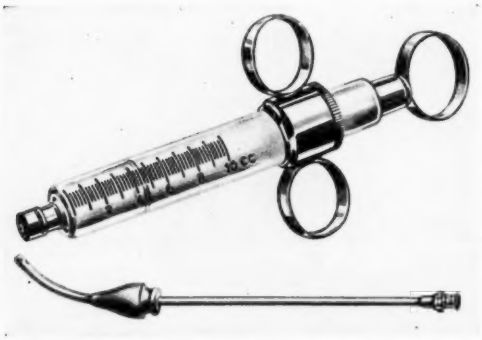

Fig. 1. Special syringe designed by the author for transuterine injections of iodized oil. A manometer attachment is being perfected.

Since my earlier paper was submitted for publication, a number of important contributions on this subject have appeared. It is my purpose to mention the more important of these articles, in order to make my review of the subject complete. More important, however, is the fact that I have observed striking therapeutic results after the use of iodized oils by transuterine injection. It is upon this phase of the subject that I propose to dwell most particularly. Furthermore, brief mention will be made of the new syringe which I now use for the injections, which has been improved by the addition of a manometer.

Recent Bibliography

The reader is referred to my previous article for a bibliographical survey of the subject of uterosalpingography. Heuser (16), of Buenos Aires, in 1925 was one of the first to employ iodized oils for gynecological diagnosis. However, since Ferré's (10) article was published about the same time as Heuser's and Forestier (12) states that Portiet, of France, was the first to inject iodized oil into the uterine cavity for the purpose of roentgen-ray study, the matter of priority is rather confusing. Heuser injected iodized oil as a means of making an early diagnosis of pregnancy in women who had missed two or more periods. Roentgenograms taken subsequently showed the normal triangular outline of the uterus when pregnancy was not present; otherwise, the oil passed around the fetus and failed to fill the entire uterus. Heuser found that the injections were in no way injurious and did not induce abortion. On the contrary, they were

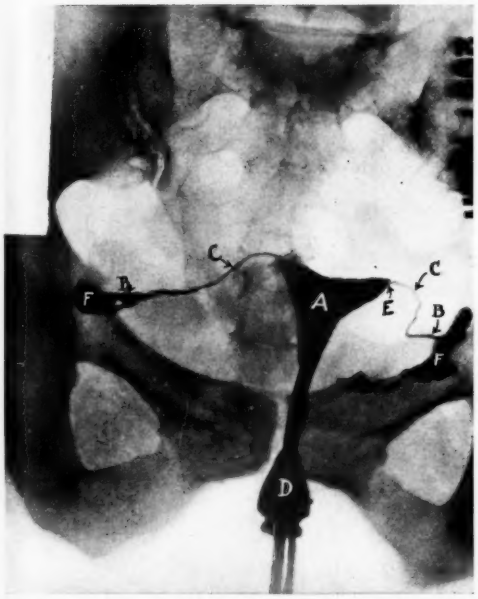

Fig. 2. Uterosalpingogram of normal genital organs. *A*, Triangular shadow of the uterine cavity; *B*, beginning of the ampulla; *C*, isthmus of the tube; *D*, nozzle and tip used for introducing oil into the uterus; *E*, snare-like compression of the tubal sphincter; *F*, free iodized oil in the peritoneal cavity.

Fig. 3. Case 1. Roentgenogram showing free distribution of iodized oil throughout the peritoneal cavity 24 hours after the transuterine injection. Such pictures may be obtained from all patients in whom the fallopian tube are patent.

ineffective when attempts were made to produce therapeutic abortions thereby.

It was not long before the diagnostic possibilities of iodized oils were recognized and their injection was used for roentgenological visualization of defects in the uterus and fallopian tubes. This subject has already been covered in my previous paper, and I shall merely add references to articles which have appeared since then.

Heuser (7), in 1926, added some important observations made with reference to a salpingo-uterine sphincter action and the muscular contractions of the fallopian tube by means of uterosalpingography. Roentgenological pictures showed at the uterine cornu and in the cavity of the tube a space similar to the ring produced in the pylorus, followed by the cavity of the tube in the shape of an ampulla. This ring is encountered in a state of relaxation or contraction, according to the phase in which it is observed. It is observed in the terminal portion of the uterine cavity, thus explaining why the fluids of this cavity are not readily transported to the pelvic cavity. This constricted ring closes in some patients when the horn of the uterine cavity becomes contracted, as seen by the shape of the walls in this region. This explains why, in the presence of uterine hæmorrhage, the blood does not enter the peritoneal cavity but escapes through the uterine cavity. The sphincter, which in roentgenograms is seen as a black line, becomes displaced with the body of the uterine cavity itself on contracting, without changing its place.

Heuser's findings not only show that the uterine cavity possesses a salpingo-uterine sphincter and that the muscular contractions of the tube extend to the uterine cavity but also indicate the different roentgenological phases in the tubal cavity as follows:

1. The iodized oil in the peritoneal cavity is absorbed by the tube through capillary attraction and expelled into the uterine cavity through muscular contraction.

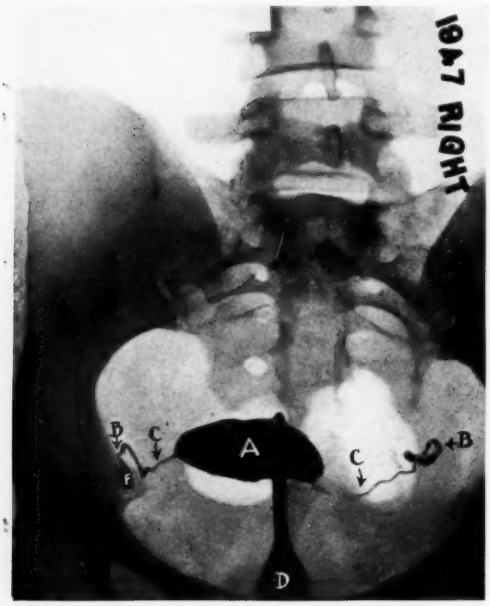

Fig. 4. Sharp anteflexion of the uterus. As the uterus is visualized, the observer faces the summit of the fundus and the nozzle points through the posterior wall of the isthmus of the uterus. For an explanation of the letters, see the legend for Figure 2.

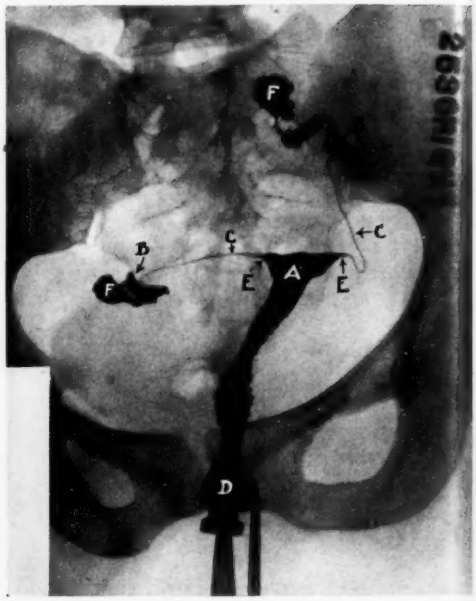

Fig. 5. Showing an unusually long, conical cervix, a displaced uterus, and a right fallopian tube situated high in the pelvis. Both of the fallopian tubes are shown to be patent. For an explanation of the letters, see the legend for Figure 2.

2. When there are remnants of placenta, the salpingo-uterine sphincter remains open in some cases thus accounting for ascending infection.

3. Remnants of placenta in the uterine cavity produce in roentgenograms a deformity of the pictures of the cavity, because of the space occupied by these remnants in the uterine cavity, thus explaining the deformities of the triangular picture of the uterine cavity when filled with iodized oil.

On fluoroscopic examination of the uterine cavity filled with iodized oil, one may observe, when the cavity is occupied by some foreign body, that muscular contractions occur as far as the neck of the uterus. They progress slowly, sometimes taking the form of undulations in the wall. In some cases, the contractions are energetic; in others, very slow. In patients with hyperthyroidism, they take an energetic course. In those cases in which there are remnants of abortions, fibromata, etc., or pregnancy, these contractions are very slow and in many cases they cannot be demonstrated.

Bakke (2), in 1927, by means of uterosalpingography, demonstrated the presence of a tubal sphincter not previously known. The tubes at the cornua are thickened in the shape of a funnel whose base is toward the uterus. This funnel is sharply defined from the uterine cavity, just like the pylorus between the stomach and the duodenum. There is a snare formation, seen in pictures of all normal tubes, which is undoubtedly produced by a sphincter. When the cornu, sphincter, and uterine end of the tube are enlarged, the resemblance to the stomach, pylorus, and duodenum is still more striking. By histological examination, Romcke (31) was able to demonstrate the anatomical substratum of this sphincteric action.

Joachimovits (19), in 1926, found the injection of iodized oils entirely harmless and of undoubted value in the diagnosis and localization of submucous and intramural uterine

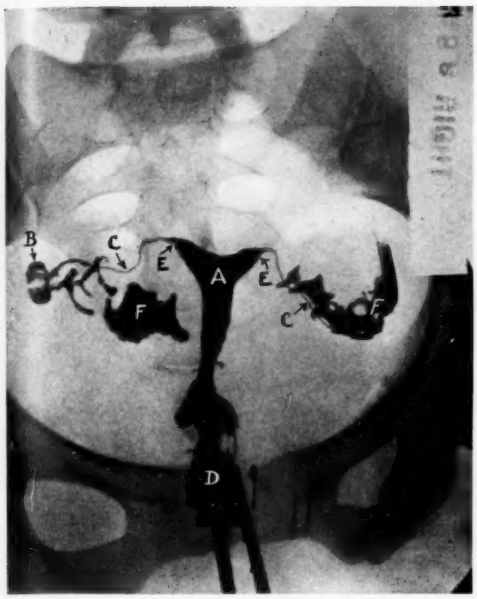

Fig. 6. The tubal sphincters, E, particularly the left, are well shown. The tubes are patent, and the oil flows freely into the peritoneal cavity. Note the large amount of iodized oil in the peritoneal cavity, *F*, although no symptoms were produced thereby. For an explanation of the letters, see the legend for Figure 2.

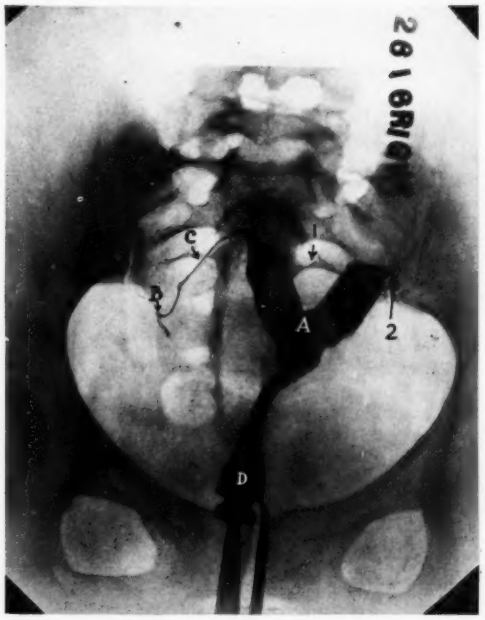

Fig. 7. Indentation, *1*, produced by uterine fibroma and occlusion, *2*, at the beginning of the isthmus. The occlusion of the right fallopian tube would have been overlooked had reliance been placed upon findings in the Rubin test. The condition shown here is frequently followed by placenta accreta.

myomata and also of intraligamentous tumors, when combined with roentgenological visualization of the ureters. By taking the roentgenograms immediately after the injection with the patient lying down, he found that contractions could be demonstrated in the normal uterus. This method of investigation makes it possible to diagnose infantilism, anteversion, retroversion, and other deviations in the position of the uterus and uterine tumors; also to demonstrate patency or occlusion of the fallopian tubes.

Cotté and Bertrand (8) in 1927, reproduced the illustrations presented with the article by Grégoire, Béclère and Darbois (14), in 1926, which illustrate the value of iodized oil injections and roentgenography in the diagnosis of various pathological conditions. For the diagnosis of tubal patency, it proved superior to insufflation; and, in the differential diagnosis of pelvic tumors, for example between myoma of the uterus and ovarian cysts, the method was very serviceable.

Nœlke (27) found uterosalpingography of special value in the diagnosis of tubal obstruction and the exact localization of the site of occlusion. He employed the method in 90 cases without admitting the patients to the hospital. For roentgenological visualization of the uterus and tubes, he first uses insufflation of air and follows this procedure by the injection of a contrast material, either sodium bromide or iodipin. Roentgenograms are made with the Bucky diaphragm. Equally good pictures were obtained with either contrast material, and no untoward effects were observed.

Béclère (4) introduces lipiodol through a soft rubber tube with an olive-shaped tip that fits the uterine cervix in each case. In order to avoid exercising too great pressure in introducing the lipiodol, he has designed a

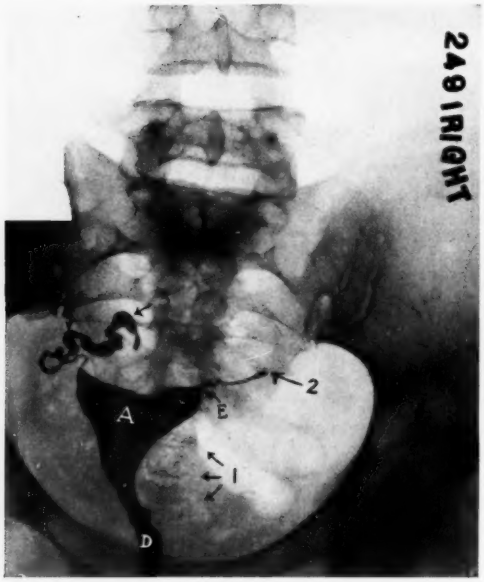

Fig. 8. Indentation, *1*, produced by uterine fibromyoma and occlusion, *2*, at the beginning of the right ampulla. *3*, Free oil in peritoneum; *4*, patent tube. For an explanation of the letters, see the legend for Figure 2.

Fig. 9. Showing a small fibromyoma, *1*, on the right side encroaching upon the uterine cavity, and a larger pedunculated fibromyoma, *2*, on the left side. The fallopian tubes are patent. For an explanation of the letters, see the legend for Figure 2.

special uterine manometer that is attached to the syringe with which the injection is made. The injection is made under fluoroscopic control, so that the iodized oil can be seen entering the tubes, and, if they are patent, passing to the peritoneum. Béclère has used this method in more than 59 examinations and has found it of special value for the diagnosis of tubal obstruction and pelvic tumors.

Contrary to most investigators, Odenthal (28) is of the opinion that uterosalpingography with various contrast materials cannot be regarded as an absolutely harmless procedure. He reports two cases in which pathological changes resulted from the injection of the contrast material. In one of these cases, 4 cubic centimeters of 40 per cent iodipin was used. There was no immediate reaction, the patient leaving the hospital 3 days after the examination and feeling entirely well; but within 10 days, she developed symptoms of peritoneal irritation with fever. After 3 weeks of treatment the symptoms subsided.

On the basis of their clinical experience with roentgenological examination of the uterus and tubes after injection of lipiodol, Grégoire, Béclère, and Darbois (15), in 1927, concluded that this procedure is practical, harmless, and of great value in diagnosis and as a guide to treatment. It shows the location, form, and volume of the uterus; also the outline of the uterine cavity and any pedunculated or other growths on its walls. It further shows the outline and caliber of the fallopian tubes, any obstruction, and its exact location. Contra-indications to its use are acute infections with fever, severe uterine hæmorrhage, and suspected pregnancy.

Grégoire, Béclère, and Darbois found that the differential diagnosis between uterine myomata and ovarian cysts, which is sometimes difficult, is simplified by the diagnostic use of iodized oil. In cases of metrorrhagia, the method shows whether or not an intrauterine tumor is present and indicates the best site for the removal of a section for biopsy.

Luettge (24) introduces 10 to 20 cubic centimeters of 10 per cent iodipin into the uterine cavity with minimal pressure. He does not induce preliminary pneumoperi-

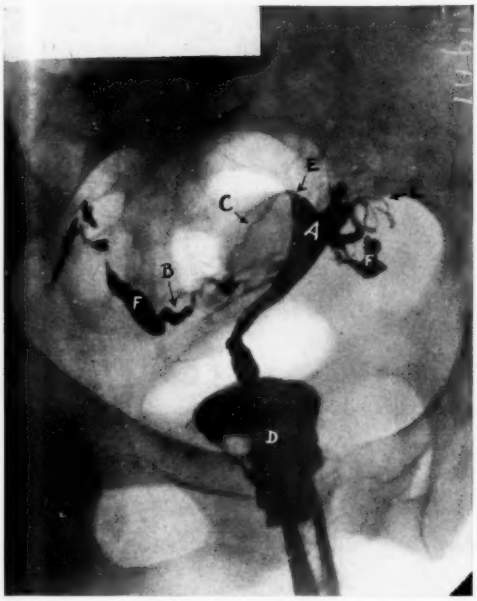

Fig. 10. Showing displacement of the uterus to the right and posteriorly. The tubal sphincter is well defined. Both tubes are patent and there is free oil in the peritoneal cavity. For an explanation of the letters, see the legend for Figure 2.

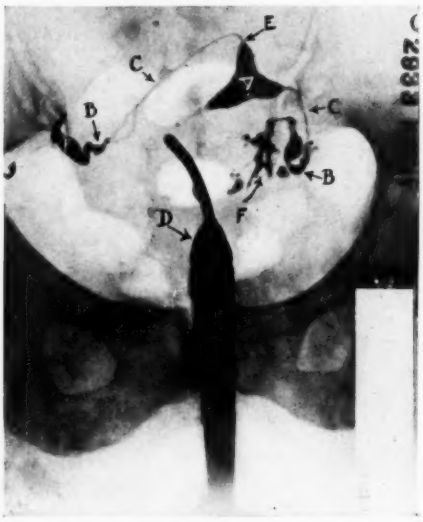

Fig. 11. Same case as in Figure 10, taken at a different angle in order to show the displacement better. For an explanation of the letters, see the legend for Figure 2.

toneum. The roentgenograms are made immediately after the injection with a Bucky diaphragm. From his experience with this method, Luettge concludes that it is of definite value in gynecological diagnosis, especially of tubal obstruction; that it causes no inflammatory reaction; and that it can be used outside of the hospital.

According to Randall (29), roentgenograms taken after the injection of iodized oils into the cavity of the uterus and fallopian tubes indicate a sharply limited field for salpingostomy in the treatment of sterility in women. He employed this method in 18 cases of closed oviducts at the Mayo Clinic. The roentgenograms indicate that in 6 of these cases, one might expect success from operative treatment for sterility so far as the condition of the tubes was concerned. Two of these patients were operated upon and in each case the pre-operative diagnosis was confirmed. One of the patients subsequently became pregnant.

Steinharter and Brown (36) found the introduction of iodized oils and roentgenography of value as an aid in the diagnosis of intra-uterine conditions, mural fibroids, patency of the fallopian tubes, the visualization of the size and position of the uterus, the recognition of early pregnancy, and in the differentiation between intra-uterine and extra-uterine tumors. They recommended it as an adjunct to the Rubin test and have found the injections to be entirely harmless.

Jungmann (22) made the roentgen-ray diagnosis of tubal pregnancy by filling the uterus in one case. The filling of the uterus showed a normal, clearly outlined, triangular area. There was no evidence of atony of the uterus, which, according to Dryoff, is present in extra-uterine pregnancy. Both tubes were completely patent. At the side of the questionable tumor, the filiform picture of the tube showed an elliptical form. The abdominal end coincided with the angle of the tube in projection. Iodized oil had entered the peritoneal cavity through the abdominal ostium, where it was visible as a flat shadow. At one point the tube was dilated to many times its normal diameter and showed a kidney-like shadow.

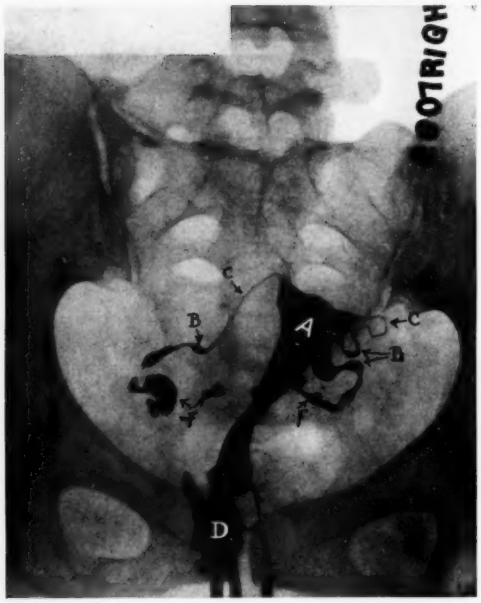

Fig. 12. Showing an adherent tube. The ampullary portion of the right fallopian tube is adherent to the posterior surface of the uterus. For an explanation of the letters, see the legend for Figure 2.

Fig. 13. Showing a double uterus with pregnancy. The right-sided uterus contains the ovum, and the iodized oil has distributed itself between the decidua capsularis and the decidua vera. The filling defect in the shadow is produced by the presence of the ovum. The mottled portion is due to the escape of oil between the villi of the decidua basalis. Note that the ovum is attached to the septum between the double uterus. Because of the poorly developed endometrium usually found in such septa, the occurrence of placenta accreta is frequent. As very little oil has entered the left-sided uterus, the latter has been outlined diagrammatically. *1*, Diagrammatic outline of other uterus not injected; *2*, shadow of oil between decidua vera and decidua capsularis; *3*, ovum; *4*, decidua basalis.

This sudden alteration in the transverse section of the tube was attributed to the penetration of iodized oil into the space between the mucous membrane of the tube and the decidua capsularis. On of the basis of this finding a diagnosis of extra-uterine pregnancy was made and confirmed at operation.

In a study of uterosalpingography in 55 cases, Reinberg and Arnstam (1 and 30) in 1926, found that, whereas the normal uterine cavity is a symmetrical triangle, the height of the triangle in the forward and backward displacement of the uterus is shortened, with the result that it assumes a spindle or segmented form. With the fluoroscope and serial roentgenograms, they observed movements of the uterus which they believed to be due to changes in the muscular tonus. In most cases they found that the uterus tends to expel the contrast material through the vagina by a sudden contraction of its walls. This muscular contraction may occur early or late, but usually 15 to 20 minutes after the injection of the contrast material. If the uterus empties itself early, this same thing occurs when another attempt to introduce the contrast material is made; and it is impossible to make a roentgenogram in such cases. There is, in addition to this method of emptying the uterus, a second method, viz., the gradual expulsion of the contrast material through the fallopian tubes into the peritoneal cavity. Peristaltic muscular contractions in the uterus could not be demonstrated with certainty.

Reinberg and Arnstam described a sphincter apparatus at the uterine end of the fallopian tube, not previously recognized. Under normal circumstances it is capable of shutting off the uterine cavity from the lumen of the tube by a contraction of the annular muscle fibers in the pars interstitialis. The width of the zone of contraction varies from 1 to 1.5 millimeters. The closure is not always complete. By serial roentgenograms, an active peristalsis of the walls of the fallopian tubes causing a rapid movement of the

Fig. 14. Uterus bicornis. For an explanation of the letters, see the legend for Figure 2.

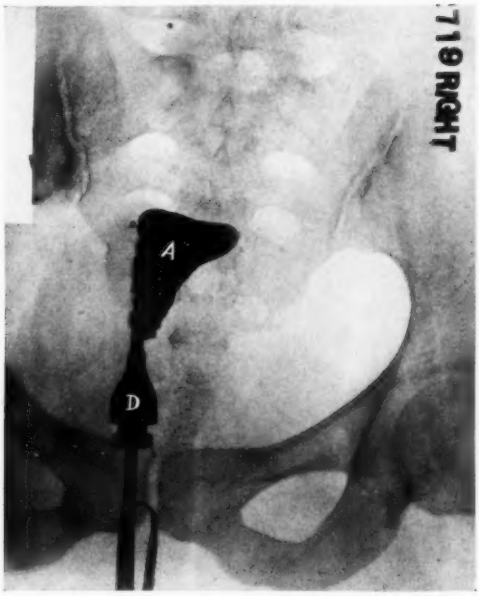

Fig. 15. Showing a complete occlusion of both tubes at the cornua. For an explanation of the letters, see the legend for Figure 2.

contents could be demonstrated. This movement was an antiperistalsis except in one instance, in which a properistalsis was demonstrated in that the uterus after expulsion of its contents into the vagina was again filled from the fallopian tubes.

With regard to the clinical applications of uterosalpingography, Reinberg and Arnstam found the measure of greatest value for the following purposes: (1) to demonstrate the patency of the fallopian tubes and localize any obstruction that may be present; (2) to differentiate uterine tumors, particularly submucous and intramural myomata; and (3) to diagnose early pregnancy, which is shown by a filling defect and a rounder shape of the uterus due to atony. They never observed an abortion due to the injection of iodized oils and believe that its careful use is permissible in selected cases.

Nahmmacher (26), used uterosalpingography in 50 gynecological cases and was able to corroborate the accuracy of the procedure by the operative diagnoses. Contrasting with the triangular shape of the normal uterine shadow, there is a clear space at the point of flexion in retroflexion. In metritis, Nahmmacher found enlargement of the uterine cavity and dilatation of the cervix. In uterine tumors there was a characteristic change in the shape and position of the uterine cavity. In ovarian tumors, on the contrary, the outline of the tubes proved to be normal. Chronic adnexal inflammation showed its presence by dilatation and stricture of the tube and finally by displacement of the uterus. Like most other workers, Nahmmacher considers uterosalpingography the best and safest means of demonstrating the fallopian tubes, and he observed no injurious effects in any of his 50 cases.

Cotté and Pièrre (9) found uterosalpingography superior to insufflation of the fallopian tubes for determining the cause of sterility, confirming the work of other investigators to the effect that this procedure not only demonstrates the presence of obstruction but also localizes the site of the occlusion. Furthermore, it indicates the nature of the operative treatment required; that is, salpingostomy, excision of the pavilion of the fallopian tubes, resection of the isthmus with implantation

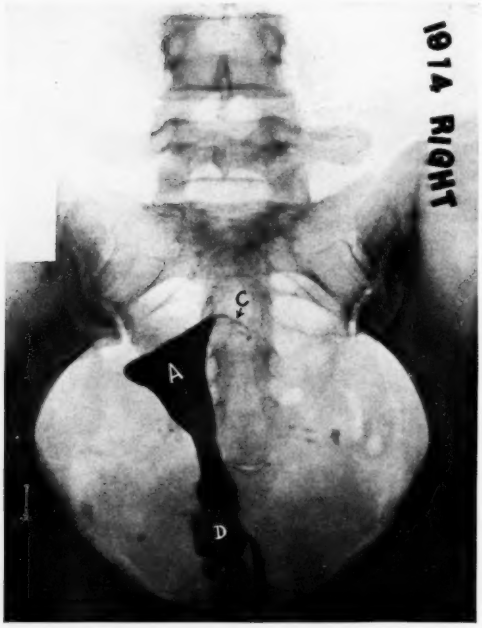

Fig. 16. Showing occlusion of the left tube at the cornu. The uterine shadow is of normal size but is displaced to the left. The left fallopian tube is occluded at the cornu. A small portion of the isthmus of the right tube is incompletely filled with oil, but the remainder of the tube is completely occluded.

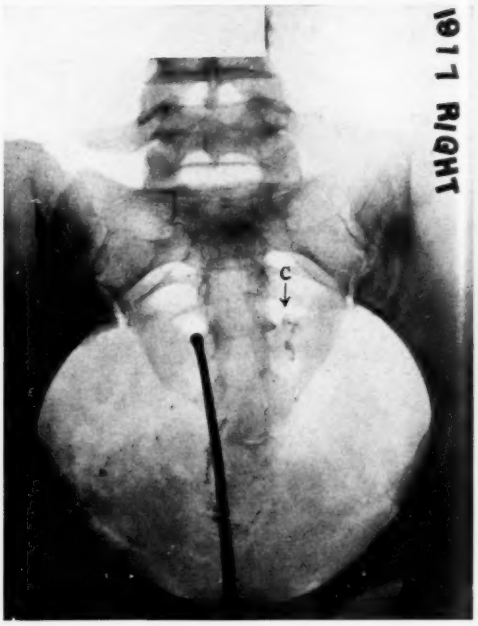

Fig. 17. The same case as in Figure 16, after the iodized oil has been allowed to drain off and a sound has been introduced to orient the position of the uterus. Note that the sound is directed to the left, corresponding to the sinistroversion shown in Figure 16. There is no shadow corresponding to the left fallopian tube, proving that the condition is really one of occlusion and not of a patent tube hidden behind the uterine shadow. The right fallopian tube shows the same incomplete filling as in the previous illustration. *C*, the isthmus of the right fallopian tube.

into the uterus, or—in cases of disease of the entire tube—transplantation of the ovary into the uterus. In the experience of Cotté and Pièrre, uterosalpingography proved especially useful for the differential diagnosis between hæmatosalpinx and ovarian cysts, as a substitute for intra-uterine exploration in cases of metrorrhagia, for the diagnosis of malformations and hypoplasia of the uterus in dysmenorrhœa and amenorrhœa, and particularly for the recognition and localization of tubal obstruction in cases of sterility.

THERAPEUTIC USE OF IODIZED OIL IN GYNECOLOGY

In my earlier work with iodized oil, my object was only to obtain roentgenograms of the lumen of the uterus and fallopian tubes for diagnostic purposes. By this means I was enabled to recognize normal fallopian tubes (Figs. 2, 4, 5, and 6), occlusion of one tube only (Figs. 7 and 8), occlusion of both tubes (Figs. 15 and 16), encroachment on the uterine cavity by neoplasms (Figs. 7, 8, and 9), uterus bicornis (Fig. 14), and septate uterus with pregnancy (Fig. 13). In cases of occlusion, moreover, the exact site of the occlusion was determined (Figs. 7, 8, 15, and 16).

However, I have been reluctant to inject iodized oil in cases of pregnancy. Although Heuser states that diagnostic injections do not interfere with normal pregnancy, I have been deterred not so much by fear of causing abortion as of doing possible injury to the offspring. In my own opinion, uterography should be employed for the early diagnosis of pregnancy only when therapeutic abortion is contemplated and injury to the offspring is of no importance.

Fig. 18.[1] Showing dilatation of the fallopian tubes with obstruction beyond the ampullæ. The fallopian tubes are obstructed beyond the ampullæ and are definitely abnormal. There is no free iodized oil in the peritoneal cavity. Twelve cubic centimeters of iodized oil were required to obtain this uterosalpingogram. For an explanation of the letters, see the legend for Figure 2.

Fig. 19. The same case as in Figure 18, with the exposure taken from the oblique position. For an explanation of the letters, see the legend for Figure 2.

Invariably before taking uterosalpingograms, I gave my patients preliminary insufflations of oxygen or carbon dioxide, if these measures had not already been employed. At the outset, I had some misgivings lest the oil might cause injury to the peritoneum or abdominal viscera or transport infectious material from the fallopian tubes into the peritoneal cavity. Subsequent experience proved that these fears were not justified.

If the objections which have been mentioned were valid, they would apply to an equal degree to the Rubin method. Yet, even though the Rubin insufflations are widely used, subsequent intra-abdominal complications are infrequent; and practically all workers are now agreed that uterosalpingography may be employed with the same measure of safety.

At first I did not suspect that injections of iodized oil might have possible therapeutic value in gynecological conditions; but I became interested in this possibility when I realized that the oil could enter the peritoneal cavity without causing injury. It was reasoned that, since an oil capable of slowly liberating iodine could be injected so as to reach the peritoneal cavity without entailing serious consequences, the use of this substance might possibly be attended with therapeutic advantages.

I was encouraged in my investigation of the therapeutic possibilities of iodized oil by the experience of H. M. Little (23), of the Montreal General Hospital, whose work I have quoted elsewhere. Little successfully used injections of 10 per cent spirits of turpentine in liquid paraffin in the treatment of infected fallopian tubes as follows: After laparotomy the pus, if present, was aspirated through a puncture in the wall of the tube and 1 to 3 cubic centimeters of the turpentine-paraffin mixture was slowly injected; the abdomen was then closed without drainage. After this treatment, Little observed no untoward effects and some of the patients subsequently gave birth to healthy children. This procedure, of course, should be attempted only after acute symptoms of adnexal inflammation have subsided.

It is perfectly plausible that an iodine-liberating preparation, proved roentgenologically to enter the fallopian tubes after intra-uterine injection, should give therapeutic results similar to those obtained by Little.

[1] Figures 18, 19, and 20 are reprinted from SURGERY, GYNECOLOGY AND OBSTETRICS.

Fig. 20. The same case as in Figure 18, taken 72 hours after injection. The roentgenogram shows that the iodized oil has gathered in the distended and closed portions of the ampullæ, *B*. Evidently there was hardly any absorption of the iodized oil from the fallopian tubes during that time.

Fig. 21. Same case as in Figure 18, taken 10 months after first uterosalpingography. This picture represents a normal uterosalpingogram. Both fallopian tubes are patent and the iodized oil is seen to enter the peritoneal cavity through the ostia abdominalia. Compare with Figures 18, 19, and 20 and note that the fallopian tubes have not only become patent but have also regained their normal contour. For an explanation of the letters, see the legend for Figure 2.

In that event, it would be obviously better to employ the treatment by this route rather than after laparotomy.

A survey of the literature on the therapeutic use of iodized oils yields very little information. Yet it will be recalled that lipiodol was originally used for therapeutic purposes, being administered by intramuscular injection for the purpose of slow iodine medication. It was only when opaque spots were noticed on roentgenograms long after such injections that Sicard and Forestier (33) in 1922, applied it to roentgen-ray diagnosis. In our enthusiasm for the diagnostic use of iodized oil, we have largely forgotten its original therapeutic application. Recently, however, Finikoff (11) has obtained excellent results in the treatment of surgical tuberculosis by intramuscular injections of sterilized peanut oil combined with a small quantity of tincture of iodine.

Sicard (32) remarks that lipiodol possesses noteworthy therapeutic properties. In his experience, rheumatic pains are considerably improved by local injections, the introduction of 5 to 10 cubic centimeters into the painful region sufficing. The injection should be deep, preferably reaching the level of the bone. Sicard obtained excellent results with this therapy in sciatica. In lumbago, sacral epidural injections worked wonders. For the articular pains of arthritis, peri-articular and sometimes intra-articular injections of lipiodol likewise proved of value.

Torto (37) believes that the subarachnoid injection of iodized oil is of value in the treatment of Pott's disease. Other than initial headache and paræsthesias in the lower limbs, he has observed no harm following this procedure. Torto is of the opinion that the direct iodine medication to the region of the diseased vertebræ should constitute ideal therapy and that the slow but continuous absorption of iodine in proximity to the lesion should be of distinct therapeutic value. However, further investigation is required on this subject.

Graevinghoff (13), in 1927, reported the use of iodipin in a case of staphylococcus infection of the ventricles of the brain with suppuration. Six cubic centimeters of iodipin were injected into the right lateral ventricle, which was markedly dilated. Several convulsions occurred immediately after the injection. This reaction was followed by definite improvement in the general condition, a drop in the

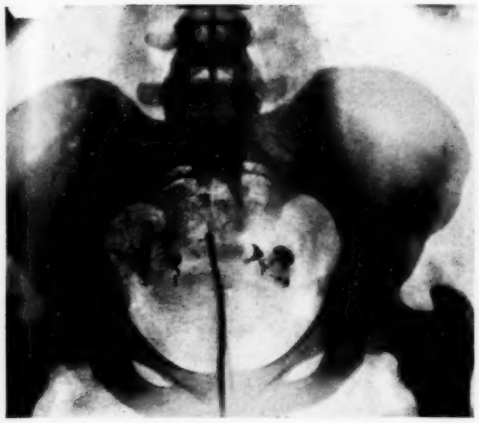

Fig. 22. The same case as in Figure 21, after the oil has been allowed to drain off and a sound introduced to orient the position of the uterus. Compare this picture with Figure 20 from the same patient, which shows tubal distention but no free oil in the peritoneal cavity 3 days after the injection. For an explanation of the letters, see the legend for Figure 2.

Fig. 23. A misleading uterosalpingogram, from which one would believe that the left fallopian tube was occluded. Compare this figure with Figure 24. For an explanation of the letters, see the legend for Figure 2. The iodized oil shows the uterus to be of normal size. The right cornu is slightly displaced to the right; the cervix, to the left. The right fallopian tube is well filled with iodized oil throughout its entire extent. The left tube is not shown. Note that there is a well defined sphincter at the right cornu.

temperature, gain in weight, and reduction in the number of convulsions. The improvement continued for 2 weeks, but after that period the child grew worse and died 25 days after the iodipin injection. Autopsy showed nearly 4 of the 6 cubic centimeters of iodipin injected still in the ventricle. The other ventricle showed inflammation of the walls, which were covered with a thick suppurative exudate; the walls of the right lateral ventricle were smooth and normal. This change in the right ventricle, Graevinghoff attributed to the action of the iodipin.

In one case of hæmorrhagic tuberculous pericarditis, Castex (5) performed a therapeutic pneumopericardium. As the roentgen-ray appearance suggested the presence of adhesions, 20 cubic centimeters of lipiodol was introduced after the withdrawal of 100 cubic centimeters of exudate. The roentgen-ray examination after this injection showed no evidence of adhesions. The lipiodol penetrated into the meshes of the inflamed tissue of the pericardium and remained there. Repeated injections were given at intervals of 5 to 14 days, but they had no definite influence on the pathological changes in the pericardium, the disease running the usual course. Injections were well tolerated. There was little or no absorption of lipiodol, as was demonstrated roentgenologically.

Many years ago, McShane (25) injected oil of sesame into the trachea and bronchi for therapeutic purposes. Jones and Jamison (21) believe that slow liberation of iodine following the injection of iodized oils into the bronchi exerts a distinctly antiseptic influence and that iodized oils are therefore of value in the treatment of lung abscess, bronchiectasis, and chronic bronchitis. Ballon and Ballon (3) note that iodized oils are beneficial in the treatment of bronchiectasis, lessening the cough and diminishing expectoration.

Cotté and Bertrand (6), in 1926, were perhaps the first to suggest the therapeutic use of iodized oils in gynecology. They observed at that time that lipiodol has a powerful antiseptic action and would destroy any microorganisms in the fallopian tube after intra-

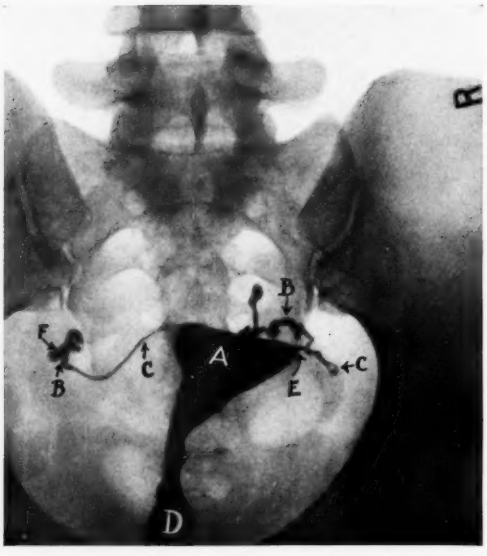

Fig. 24. The same as Figure 23, but taken from a different angle. *Both* fallopian tubes are well outlined and iodized oil has passed through both ostia abdominalia into the peritoneal cavity. This appearance proves that in Figure 23 the left fallopian tube was not really occluded but merely concealed behind the shadow of the uterus. This picture illustrates the importance of making plates from different angles. For an explanation of the letters, see the legend for Figure 2.

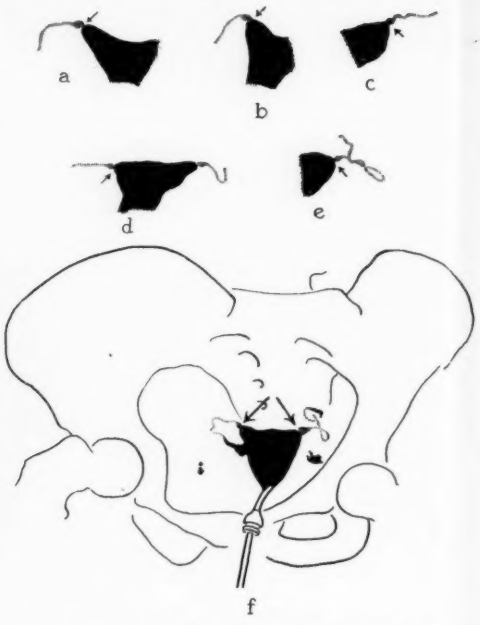

Fig. 25. The so-called tubal sphincter. Sketches drawn from previous uterosalpingograms to illustrate the action of the sphincter at the uterine cornu (a, Fig. 6; b, Fig. 10; c, Fig. 8; d, Fig. 5; e, Figs. 23 and 24; f, Fig. 21).

uterine injection. Although they used lipiodol for diagnostic purposes in more than 5,000 cases of acute or subacute inflammation of the fallopian tubes, they never noted any rise in temperature or any unfavorable reaction due to the introduction of the oil. They therefore suggested that the injection of iodized oil might prove a useful therapeutic measure early in the course of salpingitis and that it might result in early recovery and prevent tubal obstruction.

Joachimovits (19, 20) observed that in 2 cases of profuse leucorrhœa and 1 of non-gonorrhœal endometritis, there was a marked diminution of the uterine discharge after 2 intra-uterine injections of iodipin, the substance remaining in the uterine cavity for a considerable period.

Cotté and Pièrre (9) gave intra-uterine lipiodol injections in 20 cases of *acute* adnexal inflammation without harm. They believe that, in certain conditions, the iodized oil may have a favorable influence on the pathological process in the fallopian tube.

Having in mind a possible therapeutic effect from the transuterine injection of iodized oils, and also being reluctant to use force in cases of salpingitis on account of a possible danger of forcing septic material through the fimbriated extremity, I have made it a practice to inject the oil slowly and with great care. Under my direction a Luer syringe with a manometer attachment is being perfected in order that the pressure may be readily determined. A pressure of 30 to 40 millimeters is sufficient, and ordinarily the amount of material required to fill the uterus and fallopian tubes is 4 to 5 cubic centimeters. By a preliminary examination of the internal organs, one can usually estimate the amount of oil required in a given case. Furthermore, since roentgenograms are made concomitantly, fluoroscopy may be used to gauge the amount required to fill the uterus and fallopian tubes without forcing excess into peritoneal cavity.

In several of my cases of quiescent or chronic salpingitis, the iodized oil appeared to have pronounced therapeutic effect. The most striking results were obtained in Case 14, the details of which are given under the case reports. When the injection is being made for diagnostic purposes only, it is not necessary to take the pressure.

It cannot be emphasized too frequently that, if too much force is used in the injection, a spasm may occur in the musculature of the uterus and the fallopian tubes, as a result of which the patient experiences a sinking sensation or even sharp pain. Earlier in this article I have referred to the sphincteric action of the muscular bands at the uterine cornua, commonly called the "tubal sphincters"; and this function is exemplified by the findings shown in Figures 5, 6, 8, 10, 21, 23, 24, and 25. The existence of this sphincter furnishes the real reason why force should be avoided in the injection of iodized oil or the insufflation of gas. For one who has not required the sense of touch by which to gauge the amount of pressure being employed, the use of a syringe with a manometer attachment has obvious advantages.

With regard to the application of the Rubin test, one must bear in mind that the formation of a pneumoperitoneum during transuterine insufflations of gas does not give an absolute diagnosis concerning the permeability of the fallopian tubes. In Cases 5 and 6, insufflation was followed by all the evidences of free entrance of air into the peritoneal cavity. The mercury in the manometer rose only to 100 and 104 millimeters, respectively, and then dropped rapidly to 60; shoulder pain was present; and roentgenological examination established the presence of air in the peritoneal cavity. Yet the uterosalpingograms proved that only one tube was patent in each case.

In Case 8, high manometer readings reaching 210 millimeters were obtained during the insufflation of oxygen, notwithstanding the fact that several attempts were made during the same sitting. Yet, when iodized oil was injected, the fallopian tubes opened up and showed the true condition to be not obstruction of the fallopian tubes but a shortening of the right adnexa and a displacement of the uterus to the right, causing a kink in the fallopian tube. In Cases 12 and 13, the insufflation readings were high without a secondary drop; and it was the uterosalpingograms after the injection of iodized oil that located the occlusion which was at the cornual ends of the tubes.

I do not mean to belittle the value of the Rubin insufflation test, for the fact that this diagnostic procedure has been widely utilized the world over is sufficient evidence of its usefulness; but I do believe that uterosalpingograms taken after the transuterine injection of iodized oil give a more accurate and a more detailed picture of the pelvic lesion.

It is important to take several plates at different angles; otherwise, several shadows may be superimposed and thus lead to an error in diagnosis. In Figure 23, for example, the left fallopian tube appears to be occluded; but in reality it is hidden behind the uterine shadow, as shown by Figure 24. It is advisable to take one picture after the oil has been allowed to drain off from the uterus. As some oil remains in the uterus and fallopian tubes for a time, shadows of these organs will persist. On the other hand, the persistent absence of a shadow of the fallopian tube in uterosalpingograms taken when the uterus and tubes are filled with oil and again after the oil has been allowed to drain off would indicate that the oil did not enter the tube and that it is therefore occluded. The latter condition is shown in Figures 16 and 17, which indicate that the left tube was entirely occluded, while the right was only partially and incompletely filled.

In my investigation I employed lipiodol, which is a chemical compound of poppyseed oil and iodine solution.

CASE REPORTS

CASE 1. Normal uterosalpingogram. B. C., aged 35, married 11 years, complained of primary sterility. Pelvic examination revealed an anteflexed but freely movable uterus of moderate size. The cervix was directed downward and forward. The introitus was normal. The adnexa appeared to be normal. The patient received a transuterine injection of 5 cubic centimeters of iodized oil, after which the uterosalpingogram was taken. The result is shown in Figure 2, which may be taken as an illustration of

the normal genital organs. A roentgenogram taken 24 hours after the injection showed the iodized oil freely distributed throughout the peritoneal cavity (Fig. 3).

CASE 2. Sharp anteflexion of uterus. L. G., aged 25, married 3 years, complained of primary sterility. On pelvic examination the uterus was found to be sharply anteflexed but freely movable. The adnexa were not palpable. When oxygen insufflations were given, the pressure reached 200 millimeters without a drop in the mercury. Several attempts were made to force the gas through, but they were all unsuccessful so far as could be determined. There was no fall in the pressure, auscultation was negative; roentgenological examination did not show pneumoperitoneum; and the patient did not complain of shoulder pain. The patient received an injection of 4.5 cubic centimeters of iodized oil, after which several roentgenograms were taken. Notwithstanding the result of the Rubin test, the fallopian tubes were found to be patent and there was free oil in the peritoneal cavity. The typical picture of sharp anteflexion is shown in Figure 4. This is the type of case in which the Dudley operation on the cervix may effect a cure of the sterility.

CASE 3. A long, conical cervix; the right fallopian tube high in the pelvis. S. W., aged 32, married 11 years, complained of primary sterility. On pelvic examination the uterus was found to be of small size and somewhat displaced to the right. The cervix was very long and conical, with a pinhole external os. After the injection of 4 cubic centimeters of iodized oil the uterosalpingogram vividly confirmed the findings obtained on manual examination (Fig. 5).

CASE 4. Marked sphincter action at the uterine cornu. B. B., aged 22, married 2½ years, complained of primary sterility. The uterosalpingogram was taken after the injection of 8 cubic centimeters of iodized oil (Fig. 6).

CASE 5. Indentation produced by a uterine fibroma and occlusion at the beginning of the isthmus. S. L., aged 31, married 2 years, complained of primary sterility. Her menstrual periods occurred every 3 weeks and lasted 7 days. On manual examination the uterus was found to be anteflexed and slightly enlarged. There were two uterine fibromyomata: one about 5 centimeters in diameter at the fundus, its major portion apparently forming part of the uterine wall; the other, about 3 centimeters in diameter, located at the right cornu. Two days prior to the injection of iodized oil, the patient was insufflated with oxygen. As was shown by all the standard signs including the roentgenogram, the gas entered the peritoneal cavity readily and gave no indication of the existence of obstruction at the right cornu. The uterosalpingogram outlined the indentation produced by the fibromyoma at the fundus and also showed that the right isthmus was occluded (Fig. 7). Should this patient become pregnant, one may expect the development of placenta accreta, since a fibroma so located may produce an atrophy of the overlying endometrium, a condition which is often the forerunner of placenta accreta.

CASE 6. Occlusion of the fallopian tube at the beginning of the ampulla. R. D., aged 30, complained of primary sterility. Menstruation occurred every 4 weeks, was of 4 days' duration, painful, and with moderate flow. On manual examination the uterus was found to be of moderate size and freely movable. On the right side of the uterine isthmus a fibromyoma about 6 centimeters in diameter was palpated. After insufflation with oxygen, the pressure reached only 104 millimeters and dropped rapidly to 60 millimeters. It would seem from these findings that the tubes were patent. But the uterosalpingogram taken after the injection of iodized oil (Fig. 8) showed the right fallopian tube to be occluded at the beginning of the ampulla and also indicated an area of pressure from the fibromyoma on the right side of the uterus.

CASE 7. Pedunculated uterine fibromyoma encroaching upon the uterine cavity. I. S., aged 29, married 4 years, had had 2 stillbirths at term. Roentgenological study after the injection of iodized oil (Fig. 9) showed both fallopian tubes to be patent, with a small fibroma on the right side encroaching upon the uterine cavity. There was a larger pedunculated fibroma on the left side.

CASE 8. Displacement of the uterus to the right and posteriorly; well-defined sphincter at the uterine cornu. E. A., aged 34, married 6 years, complained of primary sterility. Soon after marriage, she was ill for several weeks with pelvic inflammation. Insufflation with oxygen resulted in a pressure of 210 millimeters and there was no drop. The presence of gas in the peritoneal cavity could not be demonstrated by auscultation, shoulder pain, or roentgenogram. After the transuterine injection of 6 cubic centimeters of iodized oil, the uterosalpingograms (Fig. 10 and 11) showed free oil in the peritoneal cavity, proving that both tubes were patent. They also showed the uterus to be markedly displaced to the right and backward into the hollow of the sacrum. There was a well defined sphincter in the left uterine cornu.

CASE 9. Ampullary portion of the tube adherent to the uterus. R. L., aged 31, married 7 years, gave a history of having had pelvic disease several years previously. On manual examination the uterus was found to be somewhat enlarged and to lie close to the right pelvic wall. Insufflation with 120 cubic centimeters of oxygen resulted in a pressure of 150 millimeters, dropping to 100 millimeters. The entrance of gas into the peritoneal cavity was confirmed by auscultation, reflex shoulder pain, and pneumoperitoneum roentgenologically visualized. The uterosalpingogram (Fig. 12) showed both fallopian tubes to be patent, but the ampullary portion of the right tube was adherent to the posterior surface of the uterus.

CASE 10. Double uterus with pregnancy. Mrs. F., 6-weeks pregnant, was found on manual examination

to have a double uterus. The uterosalpingogram taken after the injection of iodized oil revealed the conditions graphically (Fig. 13).

CASE 11. Uterus bicornis, S. D., aged 38, married 5 years, complained of primary sterility. After the injection of iodized oil, the uterosalpingogram showed a uterus bicornis (Fig. 14).

CASE 12. Occlusion of both tubes at the cornua. C. E., aged 30, married 2½ years, complained of primary sterility. She had suffered formerly from profuse leucorrhœa. On manual examination the uterus was found to be of moderate size, anteflexed, and movable. The adnexa were thickened. After insufflation with oxygen there was no evidence that the gas had entered the peritoneal cavity. The uterosalpingogram taken after the injection of iodized oil (Fig. 15) showed occlusion of both fallopian tubes at the cornua.

CASE 13. Occlusion of one tube at the cornu. S. G., aged 29, married 6 years, complained of primary sterility. Eighteen years previously she had had an operation for ruptured appendix; 8 years previously, one for intestinal obstruction. On manual examination the uterus was found to be moderate size, anteflexed, and sinistroverted. The adnexa were not palpated. With oxygen insufflation the pressure reached 200 millimeters, and there was no drop. After the injection of iodized oil, the uterosalpingogram (Fig. 16) showed a uterine shadow of normal size but displaced to the left.

A small portion of the isthmus of the right fallopian tube was incompletely filled with the oil, but the remainder was occluded. The left tube was occluded at the cornu. In order to remove any doubt that the left fallopian tube might be patent but hidden behind the shadow of the uterus, a second uterosalpingogram was taken after the oil was allowed to drain from the uterus (Fig. 17). This picture likewise failed to reveal any shadow referable to the left fallopian tube.

CASE 14. Therapeutic effect of iodized oil. I have referred to this case in my previous paper in SURGERY, GYNECOLOGY AND OBSTETRICS, but I am mentioning it again because I have subsequently observed a remarkable therapeutic effect following the infection. M. B., aged 34, had no children. She had had an abortion 11 years previously and was very ill at the time. Since then she had suffered from abdominal pain, which was worse at the menstrual periods. Backache was severe. Pelvic examination revealed a retroposed uterus of moderate size. The fallopian tubes were thickened and readily palpable. There was more pronounced thickening at the distal portions, giving the impression that the tubes were adherent to the lateral walls of the pelvis. The patient received an injection of iodized oil on February 8, 1927, and was subjected to roentgenological study. The roentgen-ray findings are shown in Figures 18, 19, and 20.

In November, 1927, 10 months after the first uterosalpingography, another roentgenogram was taken after injection of iodized oil. A normal uterosalpingogram was obtained (Fig. 21 and 22). Both fallopian tubes were patent and of normal contour, and free iodized oil was seen to enter the peritoneal cavity. There was also a well-defined sphincter at the uterine cornu on the right side. We cannot disregard the possibility that the injection of iodized oil 10 months previously was the essential factor in bringing about the restoration to normal conditions.

CASE 15. Misleading absence of a tubal shadow in uterosalpingogram. R. J., aged 29, married 9 years, complained of primary sterility. Soon after marriage an abdominal operation had been performed for pelvic disease. Four insufflations had been performed within a period of 2 years without obtaining entry of the gas into the peritoneal cavity. After iodized oil had been injected, the uterosalpingogram seemed to show occlusion of the left fallopian tube (Fig. 23); but, when an exposure was made from a different angle (Fig. 24), it was apparent that the left fallopian tube was not occluded but merely concealed behind the shadow of the uterus. The findings in this case emphasize the importance of making plates from different angles.

SUMMARY

1. The consensus of opinion of the great majority of investigators who have employed iodized oils for roentgenological visualization of the female genital organs is that the procedure is entirely safe and harmless and a most valuable addition to our equipment for making gynecological diagnoses. In my experience, it not only supplements but frequently supplants insufflation of gases.

2. Uterosalpingography gives us a vivid picture of the conditions existing within the female genital tract.

3. The most valuable use of uterosalpingography is for the recognition of occlusion of the fallopian tubes and the localization of the site of the obstruction.

4. In many gynecological disturbances, uterosalpingography gives us exact information that can be obtained by no other means.

5. There is a definite sphincteric action at the uterine cornu, sometimes called the tubal sphincter.

6. My experience leads me to believe that the transuterine injection of iodized oil may have definite therapeutic advantages in subacute and chronic conditions of the fallopian tubes.

7. A case is described in which uterosalpingography revealed dilatation and occlusion of both fallopian tubes but in which a

second uterosalpingography 10 months later gave an entirely normal picture. There is reason to believe that, in this case, the slow liberation of iodine within the fallopian tubes had a pronounced therapeutic effect.

8. A new syringe equipped with a manometer and specifically adapted to the transuterine injection of iodized oil is described.

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BENIGN PROSTATIC HYPERTROPHY

A Review of One Thousand Cases[1]

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EXPERIENCE in the treatment of benign prostatic obstruction during recent years has led to the development of certain principles which have contributed materially to the reduction of the mortality rate and to the safety of surgical treatment of the disease. Recognition of disease associated with prostatic obstruction, directly or indirectly the result of the obstruction, as well as recognition of disease coincident with prostatic obstruction incident to the age at which prostatic disease occurs, has served to weld the interest of the internist, urologist, and surgeon in the treatment of this condition.

Often grave organic disturbances are associated with prostatic obstruction and although obstruction may be primarily significant, the coincident organic disease must be reckoned with in the treatment of the patient. The stresses and strains of life result in degenerative changes in the cardiovascular system. The renal excretory mechanism is usually directly affected by prostatic obstruction and renal insufficiency results; however, renal function may be impaired directly by organic disease of the cardiovascular system in the absence of prostatic obstruction. Willius studied the records, including electrocardiographic data, of a large series of cases of prostatic hypertrophy and noted cardiovascular disease in 42 per cent. He concluded that the incidence of cardiovascular disease is higher with prostatic obstruction than with many other diseases during the same decades, indicating that cardiovascular disease is aggravated by persistent urinary retention.

Unquestionably renal insufficiency is a factor in decreasing cardiovascular reserve in prostatic hypertrophy. The improvement of the cardiovascular reserve coincident with improvement of renal function, following gradual decompression of the bladder and prolonged urethral or suprapubic drainage, is noteworthy in conjunction with Willius' observation on the incidence of cardiac disease in cases of prostatic hypertrophy. I have observed numerous patients in uræmic states and on the point of a cardiac break, whose cardiovascular-renal reserve has been so restored by drainage that operation could be performed safely and successfully.

It is imperative to the successful treatment of prostatic obstruction that the condition be looked on from a general medical point of view; not alone by the urologist as a disease of the urinary tract, not alone by the surgeon as a lesion requiring operation, but by the internist as a systemic disease, for there are aspects which may be best appreciated by the physician.

I have recently reviewed a series of 1000 consecutive cases of benign prostatic obstruction in which suprapubic prostatectomy was performed. The review serves as a basis for certain facts and observations presented here.

INCIDENCE AND SURGICAL INDICATIONS

True adenomatous hypertrophy comprises about 85 per cent of benign obstructing prostatic lesions, the remaining lesions being of the inflammatory or prostatitic type. While the inflammatory type is encountered often at an earlier age than adenomatous hypertrophy, there is little difference in the age incidence in cases of obstruction and retention from the two types of glands. Prostatic fullness and enlargement is found on careful digital examination of more than 50 per cent of men aged 50 years; however, the incidence of obstruction and the symptoms of benign prostatic disease are rare at that age, or under that age. The average age in this series is 64.2 years (Table I). The amount of enlargement of the gland can often be ascertained on careful digital examination. A small gland may produce intra-urethral obstruction and complete retention. On the other hand, a

[1] Read before the Western Surgical Association, Omaha, Nebraska, December 8 and 9, 1927.

TABLE I.—AGE OF ONE THOUSAND PATIENTS

Semi decades	Patients	Per cent
40–45	3	0.3
46–50	13	1.3
51–55	81	8.1
56–60	177	17.7
61–65	303	30.3
66–70	261	26.1
71–75	130	13.0
76–80	25	2.5
81–85	6	0.6
86–90	1	0.1
	1000	

Oldest patient eighty-seven.
Youngest patient forty.
98.4 per cent of patients more than fifty.
74.1 per cent of patients between fifty-six and seventy.
3.1 per cent of patients more than seventy-five.

huge intravesical gland may provide adequate urethral passage without urinary retention. The moderate-sized gland is the usual enlargement productive of retention, frequency, difficulty, and so forth (Table II). The large gland (graded 4) becomes surgical at a slightly later age than the smaller gland (Table III) indicating that even though there is very marked intravesical enlargement, mechanical obstruction may not occur early.

Experience has established the indications for operation on the basis of prostatic enlargement and residual urine. Except under the conditions of moderate or marked enlargement of the gland without urinary retention or residual urine, productive of frequency, pain, difficulty, and so forth, should prostatectomy be undertaken without reservations on the amount of benefit that may be expected? Cystoscopic examination is hardly to be recommended in all cases of prostatic obstruction; however, a small gland with no appreciable enlargement but with symptoms simulating those of prostatic disease without obstruction, usually requires cystoscopic examination. Braasch and his associates have followed the principle of employing cystoscopic examination only in those cases in which observations on digital examination do not unreservedly explain the urinary symptoms. In other words, without entering into a consideration of the symptoms and diagnosis of prostatic disease, it is essential that all such cases should be given careful urological consideration. Too often normal prostate glands have been removed for symptoms produced

TABLE II.—DEGREE OF ENLARGEMENT OF PROSTATE GLAND DETERMINED IN EIGHT HUNDRED AND SEVEN CASES BY RECTAL PALPATION, CYSTOSCOPY OR OPERATION

Enlargement	Cases	Per cent
Graded 1	79	8.7
Graded 2	391	48.5
Graded 3	274	33.9
Graded 4	63	7.8

by lesions higher in the urinary tract and readily revealed by careful examination. Retention and residual urine, when definitely attributed to the prostate, form the true indications for prostatectomy. It is questionable whether prostatectomy should be considered in the absence of residual urine or retention unless the gland is extremely large, and frequency resulting from decreased bladder capacity causes disability. Certainly, prostatectomy for the removal of a small gland not producing obstruction, but for the purpose of relieving the major symptom of frequency, is not accompanied by results which would justify the operation. Obstruction must be present to some degree before one may expect justifiable results, and usually the results are good in proportion to the degree of obstruction present.

In this series of 1000 cases, urinary retention requiring catheterization had existed in more than 50 per cent. The retention varied from one acute attack to chronic or continuous retention, requiring constant catheterization. About 23 per cent of the patients, on admission to the clinic or to the hospital, had either complete retention or were retaining urine to the full capacity of the bladder; in many instances there was involuntary leakage. This is a higher incidence than previously noted. In an earlier series of cases the incidence of complete retention was about 16 per cent. Obstruction, unless to the degree of complete retention, usually results in only a moderate amount of residual urine. In this series the amount varied from 30 to 300 cubic centimeters in approximately 66 per cent (Table IV). The size of the gland fails to serve as a criterion of the degree of obstruction (Table V) except that a higher percentage of large glands are productive of complete retention than the smaller glands.

TABLE III.—AGE OF PATIENTS AND DEGREE OF ENLARGEMENT OF PROSTATE GLAND DETERMINED IN EIGHT HUNDRED CASES

Semi decades	Grade 1	Grade 2	Grade 3	Grade 4	Cases
40–45		2	1		3
46–50	3	10			13
51–55	12	44	15	2	73
56–60	20	96	55	5	171
61–65	16	105	92	19	232
66–70	16	101	75	15	207
71–75	7	25	26	16	74
76–80	1	8	5	5	19
81–85			2	1	3
Total	75	391	271	63	800

TABLE IV.—AMOUNT OF RESIDUAL URINE IN SEVEN HUNDRED AND SIXTY CASES

Cubic centimeters	Cases	Per cent
None	31	4.0
30	79	10.3
60	81	10.6
90	62	8.1
91–150	108	14.2
151–300	147	19.3
301–450	42	5.5
451–600	21	2.7
601–750	3	....
751–900	3	....
901–1050	7	....
All urine residual	176	23.1
Total	760	

PRE-OPERATIVE TREATMENT

Experience has shown that the patient with prostatic obstruction is, as a rule, a poor subject for immediate operation. It would seem that the patient who has been about his work and who on examination appears to be in good physical condition with satisfactory cardiovascular system and little or no demonstrable renal injury resulting from moderate obstruction and urinary retention should not experience particular hazard in immediate operation. This, however, is not the case. Some time ago in a review of the relationship of preliminary treatment to mortality rate following prostatectomy, I presented data showing that the mortality rate in the apparent relatively preferred surgical risk, if operation was performed without pre-operative preparation, approached very nearly that accompanying prostatectomy after adequate pre-operative treatment in the group of cases in which the general condition was poor and there was marked renal insufficiency, infection of the urinary tract, and cardiovascular disease. The merits of preliminary treatment in all cases have been definitely established, and it may be asserted that prostatectomy should never be performed without a period of preliminary treatment. Drainage of the bladder is of prime importance, and unless there are good reasons for cystostomy, drainage should be established by a permanent indwelling urethral catheter and continued until the cardiovascular-renal reserve has been restored to the point of relative safety for operation.

During the last 3 years in the clinic, the principle of preliminary treatment in all cases for a minimum of 10 days has been carried out. A period of from 10 to 12 days of urethral catheter drainage in those patients in relatively good condition, comprising a group that was formerly operated on without preparation, has resulted in marked reduction of mortality rate. Permanent urethral catheter drainage of the bladder is the method of choice for it facilitates the one-stage visualized operation. Suprapubic drainage is necessary under the conditions of associated vesical lesions, such as stones and diverticula, and in those cases of marked renal insufficiency requiring a long period of drainage preliminary to prostatectomy. Intolerance to the urethral catheter likewise occasionally requires suprapubic drainage, but in my experience this occurs in only about 6 per cent of the patients. Suprapubic drainage as such provides an excellent method of preparation and insures the greatest safety in the management of patients in poor general condition; however, that it justifies adoption in all cases is questionable. The disadvantages of inaccurate subsequent prostatectomy places it in disfavor as a method of choice in comparison to urethral catheter drainage with the subsequent one-stage visualized operation. During the period of drainage, stabilization of renal function occurs with marked improvement in the general condition of the patient who has experienced

TABLE V.—AMOUNT OF RESIDUAL URINE AND DEGREE OF ENLARGEMENT OF THE PROSTATE GLAND IN SEVEN HUNDRED AND THIRTY-THREE CASES

Cubic centimeters	Grade 1	Grade 2	Grade 3	Grade 4	Cases
None	3	21	5	2	31
30–150	33	180	91	26	330
151–300	14	67	48	5	134
301–600	4	30	21	2	57
601–900		2	3	1	6
901–1050	1	1	3	2	7
All urine residual	13	56	76	23	168
Total	68	357	247	61	733

much depletion of organic reserve. In this series, suprapubic drainage was the method of choice or necessity in the pre-operative management in 28.8 per cent of the patients. Inasmuch as suprapubic drainage was reserved for those patients in poor general condition and those with complications, it is natural that the time between drainage and subsequent prostatectomy should be longer than if the two-stage operation were used generally. It has not seemed wise to consider prostatectomy in any case in less than 3 weeks from the cystostomy. Approximately half of the patients were operated on within an interval of a month; however, the time interval in 13 per cent of the patients was by necessity more than 100 days, and in three patients more than a year intervened between suprapubic drainage and prostatectomy.

While the general condition of the patient is the important guide to the time at which prostatectomy may be considered, the renal functional tests serve to indicate the danger of operation, rather than its safety. These tests do not serve as a means of determining renal reserve; however, a constantly low phenolsulphonephthalein return or a persistently high blood urea estimation indicates a narrow margin of renal reserve, and only under most unusual circumstances may prostatectomy be undertaken with any degree of safety if the phenolsulphonephthalein return is less than 20 per cent or if the stabilization of the blood urea is much more than 50 milligrams for each

TABLE VI.—CAUSE OF DEATH IN FIFTY-FOUR CASES

	Cases
General sepsis	15
Uræmia	10
Pyelonephritis	9
Pulmonary embolism	8
Myocarditis	5
Coronary sclerosis	2
Pneumonia	3
Bleeding duodenal ulcer	1
Peritonitis	1

100 cubic centimeters. Uræmia and pyelonephritis still occupy the position of the most important causes of death following the operation, and usually produce death when irreparable renal injury has occurred secondary to long-standing retention and infection of the urinary tract.

SURGICAL CONSIDERATIONS

Two-stage suprapubic prostatectomy is the procedure of choice and necessity under certain circumstances and provides a method of surgical procedure which, under such circumstances and in the presence of complications, carries the minimal risk. However, the one-stage visualized operation is gradually becoming more and more the one of choice and, when employed in properly selected cases after adequate pre-operative preparation, is accompanied by a minimal mortality rate and excellent functional results. In this series, the one-stage operation was performed in 72 per cent of the cases.

Certain phases of the surgical procedure are worthy of repetition and emphasis. Recent changes in the types of anæsthetic used for operations in this field have exerted a marked influence on postoperative complications and mortality. Ether, as a general anæsthetic, presents the greatest hazards in cases of prostatic obstruction, not in its administration, for there is a wider margin of safety during ether anæsthesia than in that produced by other general anæsthetics, but in the subsequent renal depression and acute pulmonary complications. The various gas anæsthetics have much to commend them; however, unless skillfully administered, they often produce cyanosis when anæsthesia is carried to the point of complete relaxation. Lundy has recently called attention to the deleterious effect

TABLE VII.—RESULTS OF OPERATION IN SIX HUNDRED AND FIFTY-SIX CASES (TWO YEARS OR MORE AFTER OPERATION)

Condition	Cases	Per cent
Practically well	357	54.4
Marked improvement	189	28.8
Slight improvement	86	13.1
No change	24	3.6
Total	656	

of repeated cyanosis. Regional anæsthesia, which has properly displaced general anæsthesia in this field of surgery, minimizes postoperative renal depression and pulmonary complications from inhalation.

The visualized one-stage operation provides exposure for accuracy in conduct of operation and application of hæmostatic measures. Accurate hæmostasis is most important. Continued postoperative bleeding rapidly reduces resistance to infection and, while few patients under modern surgical methods succumb directly from excessive loss of blood, continued bleeding exerts a profound influence on mortality through the subsequent advent of general sepsis or pyelonephritis. The Pilcher bag used in 850 cases has proved a most effective method of hæmostasis when utilized with hæmostatic control by suture at the vesical neck.

So far as mortality is concerned, it is possible to obtain an incredibly low rate for a considerable time and for a considerable number of consecutive cases either through particular good fortune or careful selection of patients, denying the benefits of operation to those patients with a narrow margin of cardiovascular-renal reserve. Accepting as contra-indications to prostatectomy only such conditions as cardiac decompensation, coronary sclerosis with evidence of marked myocardial injury, and advanced malignant hypertension, operation was performed in 140 consecutive cases after adequate pre-operative treatment with but one death. Four deaths occurred, however, in the next 54 cases, so that the mortality rate in the 194 cases in which operation was performed during that year was 2.5 per cent. On the other hand, previous to the adoption 3 years ago of the principle of pre-operative drainage in all cases, 36 per cent of the patients were operated on without pre-operative preparation with a mortality rate of 6.6 per cent. A

TABLE VIII.—SEXUAL POWER IN SEVEN HUNDRED AND TWO CASES

	Cases	Per cent
Normal before and after operation	229	32.6
Poor before and after operation	180	25.6
Normal before; poor after operation	196	27.9
Poor before; normal after operation	97	13.8

fair perspective of mortality rate may be obtained only on the basis of an average over a period of years. The mortality rate in the 1000 cases in this series (for a period of about 8 years) was 5.4 per cent and includes all deaths even though in some instances death was due to conditions apparently not related to the operation. General sepsis, uræmia, and pyelonephritis were the most important causes of death. Pulmonary embolism was the cause of death in 8 cases, coronary sclerosis in 2, pneumonia in 3 and bleeding duodenal ulcer in 1 (Table VI).

RESULTS OF OPERATION

As one must depend largely on the patient's statement regarding the symptoms of prostatic obstruction and the amount of disability produced, one must likewise depend on his statement regarding the benefit derived from treatment. The results of prostatectomy are very gratifying; about 85 per cent of patients are completely or almost completely relieved of all symptoms of prostatic disease (Tables VI and VII). A small group of patients experience no change in urinary frequency which in many instances on re-examination has been found due to pre-existing progressive pyelonephritis and infection of the urinary tract. Certainly the incidence of residual urine, and the formation of vesical calculus, and so forth, is small after complete removal of the obstructing gland.

SUMMARY

The point is emphasized that the case of benign prostatic obstruction is largely medical; a surgical condition is present, but there are aspects which may best be appreciated by the physician. Only through the combined interest and co-operation of the internist, urologist, and surgeon, can the patient be operated on with the minimal risk and assurance of the best functional result.

NEUROBLASTOMA OF THE ADRENAL IN YOUNG CHILDREN[1]

By MARTHA WOLLSTEIN, M.D., New York

PRIMARY tumors of the adrenal medulla occurring in young children, congenital in origin and malignant in course, have been known for a long time. They appear in the literature under various names. Thus Virchow (17), in 1864, decided to classify tumors of the adrenal medulla as gangliomata. Marchand (11), in 1891, described a tumor originating from the sympathetic part of the adrenal, having cells and fibrils which, on cross section, gave a granular appearance. He also noted the resemblance to round cell sarcoma. Kretz (6), in 1902, suggested that the round-cell sarcomata of the adrenal and liver reported in young children originated from the formative cells of the sympathetic system. Pepper (14), in 1901, and Hutchison (5), in 1907, published series of similar tumors under the name of suprarenal sarcoma. It was Wright (20), in 1910, who studied this group of neoplasms, collected 8 from the literature and described 4 of his own under the name of neurocytoma or neuroblastoma of the adrenal medulla. Wright drew attention to the similarity of the morphology and arrangement of the cells and fibrils in these growths to that in the anlage of the adrenal and the sympathetic nervous system at certain periods of their development. Landau (8), in 1913, used the name malignant sympathetic blastoma. Wolbach (19) preferred the name neuroblastoma sympathicum to indicate that the embryonal nerve cells composing the tumor belong to the sympathetic nervous system. On the basis of the most recent histological investigation, Bailey and Cushing (3) prefer the name sympathicoblastoma because, as they point out, not all the cells in the tumor are potentially neuroblasts, since some of them may differentiate into chromaffine cells. The sympathicoblastoma is a malignant neoplasm originating from embryonal pluripotential cells of the sympathetic nervous system. While the majority of the reported tumors of this type have been found in or growing from the adrenal medulla, they may grow from embryonal sympathetic cells or sympathicoblasts anywhere in the body. Thus tumors not involving the adrenal gland have been reported by Landau (8) from the abdominal sympathetic chain in a girl of 8 months and by Boyd (4) in a boy of 4½ years. Martius (12) described one from the cervical sympathetic chain in a boy 2½ years old. The interesting case reported by Anitschkow (2) originated in front of the lumbar spine and invaded the cauda equina. Anderson and Shennan (1) found a neuroblastoma growing in the apical region of the right lung in a girl 12 weeks old. The case reported by Pick (15) originated from the uterus, and Ritter's (16) adult cases grew from the intestine.

At the Babies' Hospital, we have accumulated a series of 9 adrenal sympathicoblastomata, proved by microscopic study of the tumor at operation or at autopsy. Three cases diagnosed clinically but not confirmed by microscopic study are not included in this series. These 9 neoplasms fall into several distinct groups clinically and anatomically.

The simplest tumor of the series has several points in common with Marchand's (11) case, briefly recorded in 1880 and minutely described in 1891.

Case 1. D. K., a female infant, presented no definite symptoms except pallor and failure to gain. She was normal at birth and was breast fed for 7 weeks. Since then she has never done well. Acute bronchitis developed and she was admitted to the hospital, where a mass "as large as the end of a man's thumb" was felt in the left kidney region. Neither kidney was palpable, and X-ray examination of the abdomen gave a negative result. The child was very feeble and died when 16 weeks old. At autopsy the acute cause of death was found to be bronchopneumonia. The left adrenal contained a globular tumor measuring 4 by 4 centimeters in diameter. It was smooth, yellow, and situated in the suprarenal medulla, covered by a thin layer of compressed suprarenal cortex about 1 millimeter thick. The tumor occupied the lower half of the gland, leaving the upper half of normal dimensions. On section the growth had an irregularly rough, trabeculated appearance. The left kidney measured

[1] From the Babies' Hospital and the Department of Diseases of Children, College of Physicians and Surgeons, Columbia University.

5½ by 2 centimeters. Its upper half was indented to receive the globular adrenal tumor. The right kidney measured 5 by 3 centimeters and was capped by a normal adrenal measuring 2 by 1 centimeter (Fig. 1). Microscopic examination showed the tumor to be divided into irregular alveoli by thin fibrous connective-tissue septa carrying blood vessels. The alveoli were filled with masses of small, round cells with deeply staining nuclei and little cytoplasm. Some mitoses were present. Between the round cells lay bundles of delicate fibrils which took the eosin stain, but not the special collagen, fibroglia, or neuroglia stains of Mallory (10) (Fig. 2). There were a number of pseudorosettes, first described by Kuester (7), where the cells were arranged around a central mass of fibrils (Fig. 3). The adrenal medulla beyond the tumor was unusually rich in round cells with hyperchromatic nuclei. The cortex of the compressed adrenal tissue surrounding the tumor was normal in arrangement, but very narrow (Fig. 2). The kidney showed no involvement in the neoplastic process. No metastases were present in any organ.

Slightly more complicated were the cases of two young infants admitted to the hospital with enlarged livers, in which nodules were palpable. The younger was only 3 weeks old, and the mother had noticed the large abdomen during the third week of life.

CASE 2. The infant was born at term, after a normal labor. The diagnosis on admission lay between birth sepsis with peritonitis and neoplasm of the liver. The child died on the second day in the hospital. At autopsy the liver weighed 690 grams; it was filled with metastatic tumor nodules, yellowish in color, with a central zone of hæmorrhage (Fig. 4). The primary growth was in the right adrenal, forming a globular mass 4½ by 3½ centimeters in diameter, cupping the right kidney. On the upper border of the tumor a small compressed piece of adrenal remained. This is a characteristic feature of the sympathicoblastomata. The growth was red, soft, and hæmorrhagic. The left kidney and adrenal were both normal. Microscopic examination showed that the primary tumor was composed of round cells aggregated into masses, with bundles of eosin-staining, delicate fibrils, ramifying between and around the cell groups. The small cells had hyperchromatic nuclei. There was a small number of larger cells with nuclei vesicular in character. A section made through the part of the adrenal gland attached to the upper border of the tumor, showed the cortical layers maintained in normal conformation, but at one point the tumor cells had invaded the reticular zone. The entire medulla was transformed into the tumor. Pseudorosettes were present. Sections of the liver metastases showed a more distinctly alveolar arrangement, with cells similar to those in the primary tumor, but fewer fibrils and larger vessels. Landau (8) has pointed out that the more

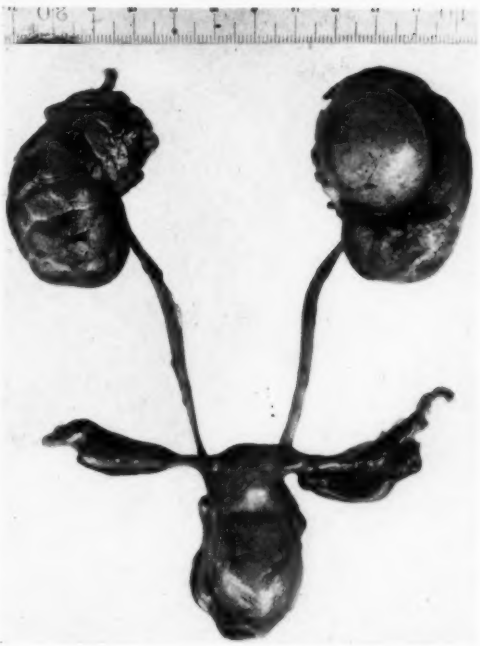

Fig. 1. Female infant, 16 weeks old. Showing sympathicoblastoma of the left adrenal cupping the kidney and an uninvolved portion of the adrenal. There were no metastases.

rapidly growing portion of these growths are less rich in fibrils. No other organs showed metastases, and the abdominal lymph nodes were not enlarged.

CASE 3. A male infant, 4 months old, was brought to the dispensary for rhinitis. The liver was found to extend 2 finger breadths below the costal margin. Five months later he was admitted to the hospital with an acute bronchopneumonia. The mother said that she had noticed distention of the abdomen the day before. At the time of admission the lower border of the liver reached the iliac crest. The child died 9 hours later, and at autopsy the liver weighed 827 grams. The whole organ was studded with round nodules varying in size from 2 millimeters to 3½ centimeters in diameter. Some of the smaller nodules were gray, but the larger ones were soft with a central zone of hæmorrhage. Little normal liver substance was left. The right kidney weighed 32 grams and was normal in size. The right adrenal weighed 2 grams and surmounted the kidney as usual. The left kidney was 7 centimeters long and was capped by a globular mass 6 by 5½ centimeters in diameter, showing on its upper pole a small flattened portion of adrenal only 1 to 3 millimeters thick. The tumor and kidney weighed 84 grams (Fig. 5). Microscopic examination gave the characteristic picture of sympathicoblastoma with pseudorosette formation.

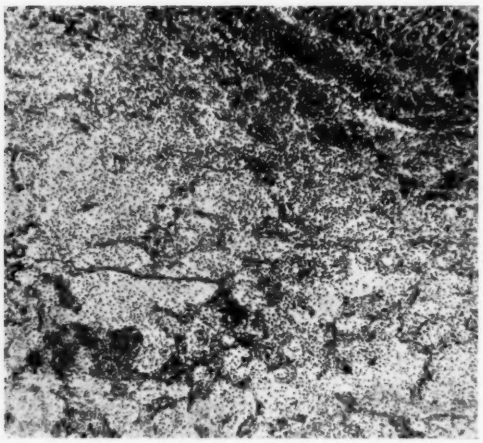

Fig. 2. A sympathicoblastoma of the adrenal medulla. The cortex is uninvolved. The architecture of the neoplasm is well shown. Low power.

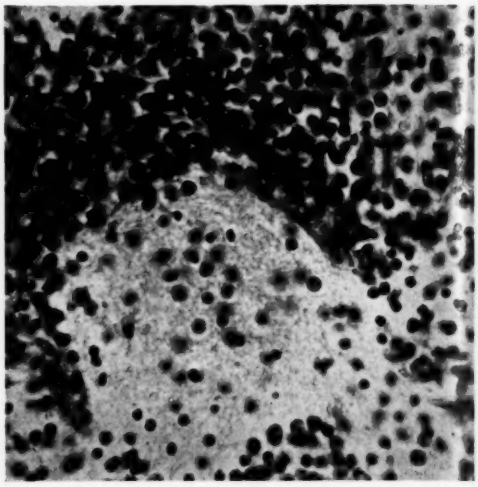

Fig. 3. Showing the sympathicoblasts and axis cylinder fibrils. High power.

The metastases in the liver showed an alveolar structure, the alveoli being filled with embryonal sympathetic cells and fewer fibrils than in the primary growth. Rosettes could not be made out in the metastases. The cell masses penetrated the liver lobules, separating and distorting the cell columns, and finally penetrating between the liver cells (Fig. 6). The right adrenal was quite normal, and the kidneys showed no neoplastic invasion. A lymph node behind the pancreas showed metastatic areas of tumor cells. The difference in the size of the liver at the age of 4 months and 9 months is striking. As an indication of its gradual enlargement is the mother's ignorance of its presence.

These two cases belong to the group which Pepper (14) described as congenital sarcoma of adrenal and liver in infants, while the two cases to be next detailed represent the Hutchison type (5) with metastases into the cranial and other bones.

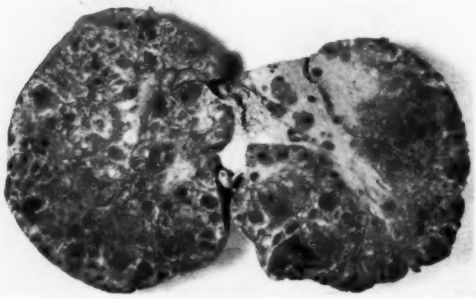

Fig. 4. Male infant, 3 weeks old. Showing the liver with metastases. Weight, 690 grams. Only a small wedge of liver substance is uninvolved.

CASE 4. E. H., a female child, 2½ years old, was admitted to the hospital with a complaint of weakness and loss of appetite. She had been a full term infant, always bottle fed, but never ill. For 3 weeks she had been fretful and had coughed. There were dark rings under the eyes suggesting old hæmorrhages. The right upper lid was yellow in color and full on palpation. The child was very anæmic, for which she was transfused but with only temporary improvement. A nodule was felt in the left lower quadrant, and grew larger throughout her stay in the hospital. Swelling over both eyes progressed, and a nodule appeared over the right parietal bone. After a month's observation the child died, and at autopsy the primary growth was found in the right adrenal gland with metastases in the left ilium, several ribs, the left temporal bone, the dura mater, the liver, the left inguinal, and the retroperitoneal lymph nodes (Fig. 7). The left temporal bone had perforated, and the dura beneath was invaded by small red tumor nodules, but the brain had not been attacked. In this and in the following case, metastases into the cranial bones appeared on the same side as the primary adrenal tumor. Microscopic examination showed that the primary tumor was a sympathicoblastoma of the adrenal, with areas of differentiated ganglion cells and nerve fibers constituting a ganglioneuroma, while the metastatic nodules were pure sympathicoblastoma. This was the most generalized case in our series, and is very similar to one reported by Monro and Dunn (13) in

Fig. 5. Male infant, 9 months old. Adrenal sympathicoblastoma cupping the kidney. The kidney and tumor weighed 84 grams.

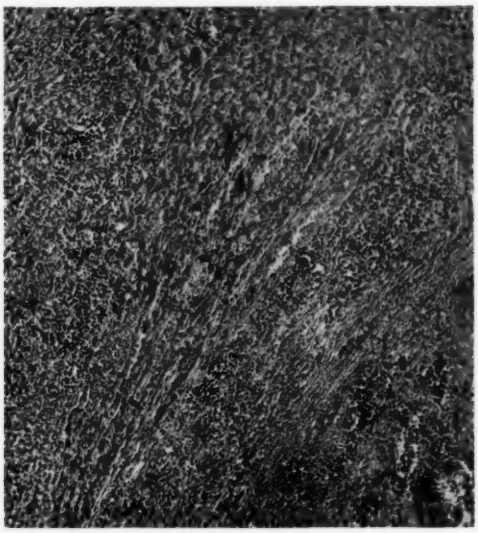

Fig. 6. The same case as that shown in Figure 5. Photomicrograph of liver metastases showing the penetration of sympathicoblasts between liver cell columns and between small groups of liver cells.

a boy 3½ years old, who died during the operative attempt to remove the orange-sized tumor in the left adrenal, which had metastasized into the bones of the skull, the ribs, and the mesenteric, retroperitoneal, mediastinal, cervical, and inguinal lymph nodes.

CASE 5. A similar but less generalized case was that of a boy of 18 months who was admitted with a history of having been pale for 2½ months, with irritability and occasional fever. He had a soft, irregular nodule behind the left ear, 5½ by 4½ centimeters in diameter. In the left upper quadrant and across the lower portion of the abdomen there was a palpable, hard mass which was not tender. Coils of intestine were felt in front of it. The liver extended 2 centimeters below the costal margin and the spleen 3 centimeters. There was a nodule over the middle portion of the left clavicle, 4 by 2 centimeters in diameter. The right knee was held rigidly in a slightly flexed position. It was swollen and tender and measured 1 centimeter more than did the left knee. Palpable lymph nodes were found in the suboccipital, posterior cervical, axillary, and right inguinal groups. The child died 3 days after admission. At autopsy the primary tumor, 9 centimeters long, with a circumference of 27 centimeters, was found in the left adrenal (Fig. 8). Just behind the pancreas a mass, 3 centimeters in diameter, surrounded the aorta. Metastatic nodules were found in the liver, the retroperitoneal lymph nodes, the twelfth left rib, and the left kidney pelvis. Microscopic examination showed that the primary tumor and the mass in front of the aorta were both sympathicoblastomata which had metastasized into the liver, bones, and lymph nodes. The mass in front of the aorta was interesting, and was found in two other cases in our series. It evidently originated from the sympathetic ganglia normally developing in that position.

A tumor originating in this region and not involving the adrenals has been reported by Landau (8) in a girl, 8 months of age. In our case the prevertebral tumor and the adrenal tumor each represented a primary focus. In Wahl's case (18) a peri-aortic mass originated from the cœliac plexus.

There were two cases in the series in which an inoperable, retroperitoneal mass was found on exploratory incision, and the origin of the neoplasm could not be determined at operation. Both these children were about 2 years of age.

CASE 6. The older was a girl of 26 months who had been born at term and had been bottle fed. The mother said that she had always had a "big, high stomach" and brought her to the hospital, because 3 weeks before she "had felt a lump in the baby's left side." This mass was found to be inoperable and the child died on the sixth day after abdominal exploration. At autopsy a retroperitoneal tumor

Fig. 7. Female child, 2½ years old. Showing the right adrenal sympathicoblastoma and ganglioneuroma with hæmorrhagic areas, cupping the kidney. There were metastases into the liver and the dura mater.

was found in the kidney region. The largest portion was on the left side, measuring 16 centimeters in length. The whole specimen consisted of the left kidney capped by a large globular tumor 7½ by 6½ centimeters in diameter, while the kidney measured 7 centimeters in length; the right kidney, freely movable, 7½ by 4½ centimeters, and between the two a nodular tumor 10 by 9 centimeters (Fig. 9). This nodular central mass showed on section the same granular, irregularly alveolar, yellowish white appearance, studded with small hæmorrhages, characteristic of, and exactly similar to, the adrenal tumor. It was notable, because unusual, that no part of the left adrenal remained attached to the growth. The masses composing the central tumor were three in number and all were globular. The vena cava passed through the mass and was slightly compressed by it. Several lymph nodes varying in size from ½ to 1 centimeter in diameter were incorporated in it. The right adrenal and kidney were unaffected. The left kidney contained a neoplastic nodule 1 by 1 centimeter in one pyramid.

In this instance it is difficult to say whether the prevertebral masses, evidently originating from abdominal sympathetic ganglia, or the growth in the left adrenal was the primary focus, and the neoplasm probably originated from two foci. Wahl interpreted his case as composed of three primary tumors.

Case 7. The other case was a boy, 23 months old who was the eleventh child. He had been breast fed for 14 months and had recovered from an attack of pertussis. For 3 weeks before admission he had complained of abdominal pain and the mother believed that the abdomen was growing larger. At the time of admission the liver extended 10 centimeters below the costal margin. The abdomen was dull in both flanks and under anæsthesia a large mass was felt and thought to be attached to the liver. Jaundice developed and the stools became clay colored. At operation, a retroperitoneal mass was found to be pressing on the under surface of the liver and causing distention of the gall bladder, which was drained. The tumor could not be removed, and the child died 7 days later. Autopsy revealed a large retroperitoneal tumor involving both adrenals and extending completely across the abdominal cavity, compressing the liver. The right kidney was embedded in the mass, but the left one was free. The tumor was almost globular and was composed of several nodules which were firm and yellowish white on section, interspersed with large red areas of hæmorrhage. The right kidney was cupped and narrowed, the tumor compressing both its upper pole and its inner border. Within its substance, however, there was no metastasis. The left kidney was normal in size and structure. No normal adrenal tissue remained. The pancreas was compressed by the tumor, and narrowed to half its normal diameter, but its substance contained no nodules. The retroperitoneal nodes were all enlarged, and some of the mesenterics as well. The spleen was in its normal position and showed no lesion. Microscopic examination showed the characteristic structure of sympathicoblastoma with pseudorosette formation (Fig. 10).

There were two cases which were more complicated in structure than 6 of the 7 others, and which, histologically, were both sympathicoblastoma and ganglioneuroma. The older of these two children was the oldest of our series, being nearly 3 years of age.

Case 8. A boy born at term and breast fed for 2 years. When he was 8 months old, the mother noticed that the right side of the abdomen was hard. He walked at 15 months and seemed quite well, but he was brought to the hospital at the age of nearly 3 years because of the "lump in the side and a limp." The mass extended from the liver to the iliac crest. Laparotomy was performed, and the right kidney with an adjacent tumor was removed. The entire mass weighed 680 grams and consisted of an elliptical, hard, smooth growth lying upon, and in front

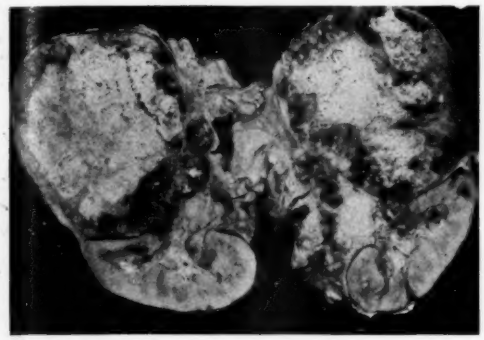

Fig. 8. Male child, 18 months old. Cross section showing the variegated appearance of the adrenal sympathicoblastoma and part of the prevertebral mass.

Fig. 9. Female child, 2 years old. Showing an inoperable, lobulated, penetrated mass and large, globular sympathicoblastoma in the left adrenal.

of, the right kidney, which was entirely separate from the tumor and normal in shape and structure. The tumor was 18 centimeters long and 11 centimeters wide, and its circumferences were 37 and 27 centimeters respectively. On section the lower part of the tumor was found to be red, mottled, soft, areolar in appearance, showing many yellow points of calcification, some so hard that they grated against the knife. The upper third was white, areolar, and firm, and contrasted with the softer, embryonal appearance of the lower two-thirds. The microscopic diagnosis was ganglioneuroma of the adrenal. The child recovered from the operation and could not be traced for final report.

CASE 9. The other patient was a boy, 15 months old, whose parents had been treated for syphilis 5 years before his birth. There had been 11 pregnancies, and only 3 children were alive. The patient was born at term and was nursed for 9 months. He was brought to the dispensary because he was irritable, sleepless, and underweight. His Wassermann reaction was negative. A hard, globular mass was felt in the abdomen in the left upper quadrant. The liver was palpable. Axillary, inguinal, and posterior cervical lymph nodes were palpable. The spleen was felt above the mass and reached only to the costal border. The urine was negative; the blood showed a moderate anæmia. Operation was performed by Dr. Bolling and a large mass was removed with the diagnosis of adrenal neoplasm. Dr. Bolling's notes read as follows: "An irregular nodular mass lying above but not adherent to the left kidney, retroperitoneal. There were no enlarged glands nor was there any evidence of extracapsular growth beyond the midline. The peritoneum lateral to the descending colon was incised and the tumor delivered with considerable difficulty, partly by blunt dissection and partly by isolating adhering areas containing bleeding vessels and cutting between ligatures. There was no connection with the kidney. After removal of the tumor, no attempt was made to suture the incision in the peritoneum. The wound was closed in layers with three retention sutures. The child's condition was fair throughout."

The specimen was an elliptical mass weighing 140 grams. It measured 9 by 7 by 3½ centimeters. The surface was irregularly nodular, and the capsule roughened but intact. Adherent to the posterior surface was a much compressed piece of adrenal gland 5 centimeters long, 2 centimeters wide, and 2 millimeters thick (Fig. 11). It was possible to dissect this portion of adrenal free, so that both surfaces were seen to be flattened, and on section the cortex and medulla were only a line in thickness. Its upper end merged into the tumor, and could not be separated from it. The cut surface showed that the neoplasm was composed of small, round lobules varying in size from 3 millimeters to 1 centimeter in width. These were either pink or dark red in color, with yellow areas of inspissated blood. At the upper pole of the tumor, there was a pink, even, smooth, dense area 2 centimeters wide and 3½ centimeters long, which had the smooth appearance of a sarcoma, quite in contrast to the variegated appearance of the rest of the growth.

Microscopic examination of a section from the upper pole of the tumor including the dense (pink) area, showed that the structure of the soft central portion differed from that of the periphery. In the former were masses of cells varying in size and shape, arranged in smaller and larger alveoli bounded by cellular fibrous tissue. The cells were partly round and small, partly larger and multinucleated, and partly triangular in shape; some of them had nuclei at either pole and none showed mitotic figures. Between the cells were masses of delicate fibrils, some of which were connected with the cells. Definite pseudorosettes were seen. The blood vessels were small and numerous, carried by the connective tissue between the alveoli. In the dense pink layer at the periphery of the tumor, large cells like adult ganglion cells were numerous (Fig. 12), while masses of nerve fibers having a definite neurolemma formed

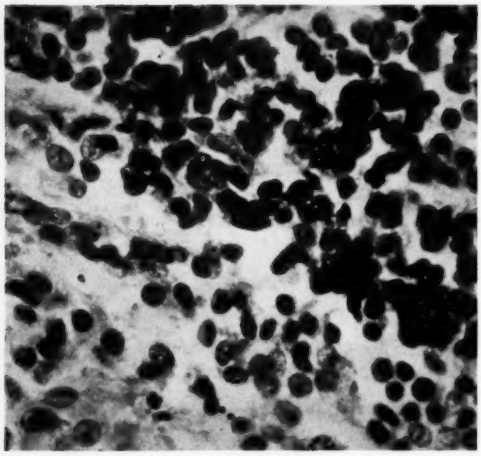

Fig. 10. Photomicrograph showing the sympathicoblasts and pseudorosettes. High power.

a wide layer, like a capsule. The ganglion cells occurred singly, in columns and in groups, as in a ganglion. But there were also masses of the embryonal nerve cells in this, the densest portion of the tumor. It was apparent that the neoplasm was both a sympathicoblastoma and a ganglioneuroma; that is, it contained areas of embryonal and of adult nerve cells, both originating from the pluripotential sympathicoblasts of the adrenal medulla. This boy is alive one year and 5 months after operation.

Lehmann (9) reported the case of a boy who was well 2½ months after operation, performed when he was 11 months old. The child operated upon by Dr. Bolling is the only one recorded who was alive one year after operation.

In our group of 9 cases of neuroblastoma of the adrenal medulla there were 3 boys and 6 girls. The tumor involved the left adrenal in 4 cases, the right in 4, and both adrenals once. The age of the children varied from 3 weeks to almost 3 years, 3 being under the age of 1 year, 3 in the second year, 3 in the third year. There are 5 different types in this series, according to the location of the primary tumor and of its metastases. Thus we have first the simplest case of the series in which a small nodule was found in the left adrenal, leaving the greater portion of the organ in an apparently normal condition and forming no metastases. This tumor showed microscopically the most typical and exquisite

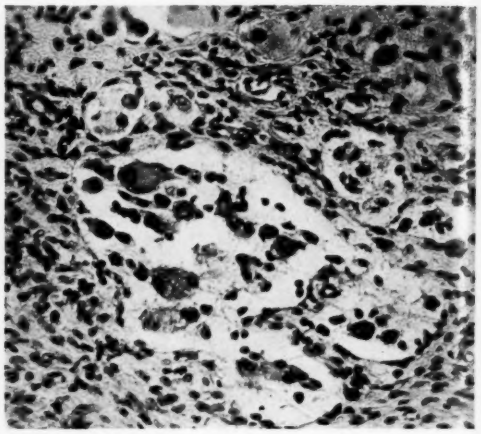

Fig. 12. Ganglion cells in Case 9.

structure of embryonal sympathetic cells and fibrils according to Wright's classical picture.

The next type is illustrated by 2 cases, both in young infants, 3 weeks and 9 months old, respectively, and both boys. The younger child showed the tumor on the right side, the older one, on the left side. Both developed metastases in the liver, enlargement becoming so great that it was the outstanding symptom. No other metastases were present. While cases of this type occur most often in young infants, Wolbach and Morse (19) have reported a case in a boy 4 years old.

The third type conforms to what has been known as the Hutchison type (5) of round-cell sarcoma of the adrenal. It is represented in our series by 2 cases, 1 boy and 1 girl, 18 months and 31 months old respectively. In the latter it was the right adrenal which was affected, while in the former the left side was the seat of the primary growth. Both showed metastases over a much wider area, involving bones of the skull, the ribs, the pelvis, the clavicle, and the liver, and in the case of the girl, the dura mater. The liver was involved to a much less extent than was the case in the second group. The children thus affected are more often in the second or third year of life. In spite of the differentiation of part of the growth into a ganglioneuroma, metastases of sympathicoblastoma were present when the child was first seen.

In the fourth group there are again 2 patients, a boy of 23 months, and a girl of 26 months. In the boy, both adrenals were affected; in the girl, only the left. Both of these cases were characterized by an inoperable tumor lying in front of the vertebral column between the adrenals and kidneys, making a mass from which kidneys, adrenals, and nodes had to be painstakingly separated and dissected. In both children the prevertebral mass represented a second tumor focus. Landau's (8) case of an 8-months-old girl had a neuroblastoma primary in the retroperitoneal lumbar region which metastasized into the liver. In our case there was a metastasis in the kidney on the same side as the large tumor in the adrenal. The right-sided tumor compressed the liver, but did not invade it and jaundice developed because of the mechanical effect of the tumor on the common duct.

The final group was the most interesting of all, and comprised 2 tumors, 1 from the right adrenal of a 3-year-old boy and the other from the left adrenal of a boy, 15 months old. Both were removed by operation, and the younger child is alive 1 year and 5 months later. It is characteristic of both of these tumors that they were entirely separate from the kidney, which was not compressed or deformed by the growth. In all the 7 other cases, the upper pole of the kidney was cupped to receive the globular adrenal neoplasm. In the last 2 cases the neoplasm was also globular, but it was entirely separate from the kidney, and readily removable without it. In both cases a small, much compressed portion of the adrenal remained on the upper pole of the neoplasm, and this portion of the adrenal gave a normal histological appearance. It is characteristic of all the adrenal tumors that their cut section has a granular, irregular alveolar appearance, described by Wahl (18) as variegated, and unlike the soft, brain-like consistency of the embryonal kidney sarcomata.

The tumors of the fifth group showed two distinct portions on section. One was pale pink, very firm and even in appearance, while the other had the granular, roughened appearance characteristic of all adrenal neuroblastomata. Microscopic examination showed that the firmer portion was characterized by the presence of many large, adult nerve cells with distinct nuclei and nucleoli, while the other portion of the tumor was composed almost entirely of the embryonal type of sympathetic nerve cells with numerous fibrils representing axis-cylinders. Chromaffin cells were not present, and both tumors were classified as neuroblastoma and ganglioneuroma. Martius' tumor of the cervical sympathetic, while chiefly a neuroblastoma, presented one firm nodule which was a ganglioneuroma. Wahl's tumor, in a girl, 2½ years old, contained areas of neuroblastoma, ganglioneuroma and chromaffin tumor, all being variously differentiated stages of the same embryonal, pluripotential cell.

Fig. 11. Male child, 15 months old. The adrenal sympathicoblastoma and ganglioneuroma was removed by Dr. Bolling. The child is alive 1 year later.

Clinically the most rapidly fatal cases are those which present the large liver as the main symptom. The tumors which metastasize to the bones often present swelling and discoloration of the lids of one or both eyes as the first symptom, the adrenal primary tumor being found on abdominal palpation. The sympathicoblastomata originating in the adrenals and in the abdominal sympathetic ganglia, resulting in a prevertebral mass

between the adrenals and kidneys, without metastases, cannot be diagnosed with any degree of certainty. Those rare sympathicoblastomata of the adrenals which are not adherent to the kidney and show areas of differentiated ganglion cells may be operable, because they apparently do not metastasize early. They contain areas of neuroganglioma as well as of sympathicoblastoma, and it is the latter only, apparently, which forms metastases.

CONCLUSIONS

In a series of 9 sympathicoblastomata of the adrenal medulla in young children, 3 contained areas of ganglioneuroma in parts of the tumor. One such tumor metastasized very generally to the bones, dura, and liver. The 2 others were removed by operation before metastases occurred. Both children recovered from the operation and one is alive a year and 5 months afterward, the only one recorded to survive so long.

In 7 of the 9, the tumor proved inoperable when first diagnosed.

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THE INSULIN-GLUCOSE TREATMENT OF TRAUMATIC SHOCK

A Discussion with an Experimental Report

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MUCH evidence has accumulated, especially during the World War, that indicates that the chief factor in the production of traumatic shock is a toxin developed in damaged tissue (1, 2, 4, and 11). The toxic agent is unknown, but histamine is an example of a protein cleavage product which produces experimentally the classic picture of shock (9). It is probable, however, that histamine does not represent the only example of a protein autolytic toxin which will produce shock-like symptoms. The toxins liberated as a result of bacterial activity have been suggested as being of a somewhat similar nature. Anaphylactic shock is probably an additional example of a similar protein cleavage product producing certain symptoms sometimes similar to those of shock (10).

DEFINITION OF TRAUMATIC SHOCK

Cannon (4) in his monograph on "Traumatic Shock," defines the condition as follows: "It is characterized by a low venous pressure, a low or falling arterial pressure, a rapid thready pulse, a diminished blood volume, a normal or increased erythrocyte count and hæmoglobin percentage in peripheral blood (thereby differing from simple hæmorrhage), a leucocytosis and an increased blood nitrogen, a reduced blood alkali, a lowered metabolism, a subnormal temperature, a cold skin moist with sweat, a pallid or grayish or slightly cyanotic appearance; also by thirst, by shallow and rapid respiration, often by vomiting and restlessness, by anxiety changing usually to mental dullness and lessened sensitivity."

TERMINOLOGY

Cowell (7 and 8) divided traumatic shock into primary and secondary wound shock. The former condition supervenes immediately when the damage sustained by the body is great and death often results unless prompt treatment is possible. Primary shock is much less common than secondary traumatic shock. The latter is the usual shock state which frequently appears after a lapse of several hours. In primary shock the symptoms appear to be the result of a powerful stimulus of nervous tissue which results in a reflex relaxation of the vascular tree. It has been suggested that such shock is more likely to occur in the "highstrung" or in those with temperamental instability. It may be similar in nature to the conditions initiating fainting or syncope but persists somewhat longer (4 and 18). Modern evidence seems to prove that secondary shock is initiated and due principally to a toxic factor, which increases the permeability of the capillary walls resulting in a decreased blood volume and a falling blood pressure. Because of the rapid loss of blood volume through the capillaries, Cannon suggests the term "exæmia" as being descriptive of the essential state of the circulatory apparatus in shock.

CONTRIBUTORY FACTORS IN SECONDARY SHOCK

Many observers have emphasized the fact that the toxic agent actually seldom works alone. Among initiating factors (18) there is often some blood loss. Infection may increase the potency of the toxic agent. Likewise, exposure to cold, because of the tendency to lowered metabolism and lowered temperature of the body, has been found a very important contributory factor. Lack of food and water or water loss by any method whatsoever, as perspiration, purging, and vomiting, may play a rôle in the reduction of the blood volume (4). Baruch (2) found the oxygen-carrying capacity of the hæmoglobin lowered. This fact, plus a decreased volume flow, a lowered blood pressure, and an increased blood viscosity (4), handicaps the internal cellular respiration. An anæsthetic, such as chloroform or ether, adds to the disturbed metabolism

both before the blood pressure falls and more so afterward. Thus, note is to be made of the fact that, although a liberated toxin in traumatic shock is usually the chief factor, the final condition of the patient is the result of a group of factors. Therefore, improvement may follow when some easily controllable condition is eliminated, as for example cold.

SUSTAINING FACTORS IN SECONDARY TRAUMATIC SHOCK

After the occurrence of the initiating factors of secondary shock, certain sustaining factors become operative and should receive consideration (18). Chief among these are the low blood pressure, a decrease in blood volume, a fall in body temperature, and a reduction of metabolism (4). As a result of these changes there is a decrease in the supply of oxygen to the tissues with a tendency toward disintegration (18). Theoretically, therefore, provided the factors just mentioned continue to be operative, the injury to the capillary endothelium becomes more marked, and the exudation of the blood plasma continues, causing a progressive reduction of the volume of the available circulating fluid of the body. As Cannon has said, a series of "vicious circles" are probably started, dependent upon retarded blood flow, lowered metabolism, and increased blood viscosity. Krogh (23) has shown that an anoxæmia produces capillary relaxation, an event which of itself would seem capable of decreasing the available circulating fluid of the body without the aid of the progressive exudation of the blood plasma which seems to be so important as a final factor in secondary shock. All of these deranging processes take an appreciable period of time for their operation and finally end in death unless the "vicious circles" are broken.

RELATION OF SURGICAL TO TRAUMATIC SHOCK

In surgical shock, as we see it in ordinary hospital work, hæmorrhage, toxic agents from infection, the harmful effect of anæsthesia (especially if it be ether or chloroform), and loss of water from the body by sweating or preparatory purging, all may be more or less operative. Dale (9 and 11) in his work on histamine shock, has shown that in the unanæsthetized animal 10 milligrams of histamine per kilo is without marked effect, while on the other hand etherization with open mask for 2 hours renders the animal very susceptible to small doses of this toxin. Ether alone may act in such a way as to increase the capillary permeability (4). A somewhat lowered blood pressure is a result to be expected (30) when the effects of ether are added to those of tissue damage. The larger the amount of tissue damage, the greater the likelihood of definite shock symptoms.

In abdominal operations as soon as the intestines are handled, there is a vasodilation with distention of the capillaries and veins, due possibly to local inhibition of vasomotor tone, to a change in intra-abdominal pressure, to venous obstruction, or possibly to all of these factors working together. The stasis or lessened return of available fluids to the circulatory tree, due to such sequestration of blood during and after abdominal surgery, even without blood loss, may occasionally explain the occurrence of shock-like symptoms. It is obvious that such a vasodilatation of the portal vessels decreases the available blood volume and interferes with the return of blood to the heart, which in turn decreases the blood pressure and the volume flow of blood to the outlying tissue. Erlanger and Gasser (14) produced shock experimentally by retarding the return of venous blood to the heart. Thus, traumatic shock and shock following abdominal operations may be very much alike although the initiating factors and sustaining factors may differ somewhat.

OUTLINE OF SHOCK TREATMENT

Because of the vicious circles which are soon established, the treatment of severe shock has not been satisfactory. The obvious points of attack have been first to correct as quickly as possible all of the contributory or initiating factors by giving rest, opiates, applying heat, stopping fluid loss, and preventing if possible toxic absorption from damaged tissue. The absorption of toxic protein products from damaged tissue has been prevented by excision of such tissue. The sustaining factors have been attacked by attempting to increase the

blood volume, the blood pressure, and the volume flow of oxygenated blood to the outlying tissues, and also by preventing a continuation of the toxic absorption.

As a preventive measure in shock, Cornioley and Kotzareff (6) have attempted to immunize animals against the shock toxin by previous injection of blood and serum of shock animals, but without conclusive results.

Crystalloid solutions such as salt solution, Ringer's solution, and glucose solution have been tried repeatedly, both in isotonic and hypertonic strengths. During the war, colloidal solutions such as gum-acacia (3) were added to crystalloid solutions with results not particularly satisfactory. Sodium bicarbonate was given a trial by Cannon, Fraser, and Crowell (5) but without good effect. Blood transfusion given slowly seems at present to be the best intravenous treatment, although its value is by no means certain, especially in extreme cases. Hanzlik and his collaborators (22) have given some evidence that citrated blood is harmful to the organism in concentrated solutions, causing certain definite histological changes in the lungs. Considerable evidence is accumulating that our common intravenous preparation which we have used so freely, and often without entire knowledge of the actual effect it was having on the blood corpuscles, plasma, and the tissue of the body in general, may be sometimes actually harmful (12, 13, 20, 21, 22, 24).

Robertson and Bock (29) have given some interesting evidence that in cases of reduced blood volume, fluids administered by way of the alimentary tract are to a high degree retained in the circulation. The remarkable feature of their results is the increased volume of fluid held in the blood vessels when water and salt solutions enter the body by natural channels as compared to injected solutions.

Theoretically, crystalloids, such as glucose and salt, even when introduced slowly and in hypertonic strengths, would be expected to cause only a temporary rise in blood pressure unless the vicious circles of shock are in some way interrupted by the introduction. The crystalloid molecule (28) is smaller than the colloid molecule and has a higher osmotic pressure. It might, therefore, be expected to pass readily out of the circulation since the damaged capillary walls in shock are already permitting the passage of the larger molecules of the colloidal blood plasma. Immediately after the introduction of a crystalloid solution, because of its relatively high osmotic pressure, some fluid may be drawn from the tissue spaces into the circulation. However, the small crystalloid molecules very quickly pass through the capillary wall, and an osmotic balance is struck between the outside of the capillary wall and the inside, after which substantially the same condition exists as before treatment. Even if the crystalloid did not strike an "inside outside balance," so to speak, it would probably pass off through the kidneys as crystalloids will pass through the kidneys as long as the blood pressure is above 40 millimeters of mercury. Thus, crystalloid solutions, including glucose and chloride solutions, have generally been ineffective in the treatment of shock.

Although the osmotic pressure of a colloidal solution is not as high as that of a crystalloid solution, the larger molecules of the former might find difficulty in passing out through the interstices of the capillary endothelium and thereby aid in maintaining the blood volume and pressure. The use of gum-salt solution of Bayliss (3), and the glucose-acacia solution of Erlanger (14) are attempts to hold fluid in the circulatory tree by combining the high osmotic pressure of a crystalloid with the large molecule of an innocuous colloid. Bayliss, as late as 1922, warmly defended his solution as having some value, and other favorable reports of its usefulness have appeared (15). Clinical experience with the solution of Bayliss and the solution of Erlanger has not been entirely satisfactory, and there have been reported fatalities (25 and 26) due to these solutions. Certainly, on the whole, the hope held out originally for solutions containing mixtures of crystalloids and colloids has not received universal encouragement.

More recently Kruse (24) and Hanzlik and Karsner (21) have presented new evidence which now must be taken into consideration when any intravenous solution is used. Hanzlik and his associates have found that gum-acacia apparently produces rather definite

physical and chemical changes in the blood. Among the changes that may occur after the intravenous injection of such colloids as gum-acacia and various hypertonic solutions of crystalloids in the concentrations that have been used in clinical work, are hæmagglutination, hæmolysis, darkening, and rapid sedimentation of the blood, lowering of the hydrogen ion concentration, increase of lactic acid, and a diminution in carbon dioxide content of the blood. Of obvious striking importance were changes observed in the hæmoglobin and red blood corpuscles followed sometimes by the occurrence of minute pulmonary emboli with capillary thrombosis and extracapillary hæmorrhage and œdema. Pulse, blood pressure, and respiration changes were also apparent. More recently Applemans (1) has compared the effect of 6 per cent acacia on the viscosity of oxalated blood and sedimentation of corpuscles *in vitro* with the changes in these physical factors of the blood after intravenous injections in rabbits. He confirms alterations in the blood as found by Hanzlik. The viscosity of the blood is increased. At times the blood was so thick that its viscosity could not be measured. The rapidity of corpuscular sedimentation was greatly increased. Control solutions of sodium chloride, however, produced no such effect. Finally, Hanzlik and Karsner have shown that many agents, among which was acacia in the concentration which has been used in the treatment of shock and hæmorrhage, agglutinated red blood cells *in vitro*, indicating a basis for preliminary embolic and other histological evidence of lung injury. Foster and Whipple (17) have noted that intravenous injection of acacia delayed return of the fibrin content of the blood to its normal value and that clotting is delayed or prevented. The latter fact is of importance when contemplating the use of gum-acacia solutions in uncontrolled hæmorrhage. Gessell (19) warns that the greatest care should be used in the use of blood substitutes in man. In his experience, toxic effects, with a fall in blood pressure, were not an unusual occurrence.

GLUCOSE-INSULIN IN SHOCK

In a recent paper, Fisher (16) has advocated the use of glucose and insulin in the treatment of shock. His conception of shock and its rational treatment is based upon the following statement: "Our conception of shock is that in this state an internal asphyxia and acidosis with oxidation processes held in check or abeyance result in a state of exhaustion. I personally believe that this state is primarily caused by a sudden derangement of the central nervous system. Whether these two conceptions are correct or not, any method promoting combustion and oxidation, and at the same time furnishing heat energy, should be effective in combating shock." Fisher suggested that insulin might serve to increase the rate of oxidation of glucose in the tissues and aid in supplying carbohydrate or food and oxygen in some manner to the injured and anoxæmic cells—thus aiding in combating the injurious effects of the toxin which is thought to produce shock. After a review of the literature concerning our present knowledge of the fate of glucose after insulin injection, no reason was obvious to us why glucose-insulin should act more beneficially in shock than a hypertonic solution of the crystalloid glucose, without the addition of insulin. Fisher mentions 31 cases of shock treated successfully with glucose and insulin and records in detail 5 typical cases. Two of these cases he regards as toxic or septic shock. We wonder if such cases should be classified with traumatic shock since they present factors not found in this condition.

In an effort to aid in establishing the true value of glucose and insulin in the treatment of traumatic shock we have carried out a series of experiments upon dogs.

EXPERIMENTAL RESULTS

Each dog was anæsthetized with ether. After the first few minutes of anæsthesia, very little ether was required to keep the animal quiet. The carotid artery was connected with a mercury manometer and the blood pressure readings were recorded on kymograph paper. Shock was produced by hammering the thigh muscles of one leg followed by occasional massage until the blood pressure fell below 70 millimeters of mercury with a definite tendency for the fall to continue. After this state was produced treatment was begun.

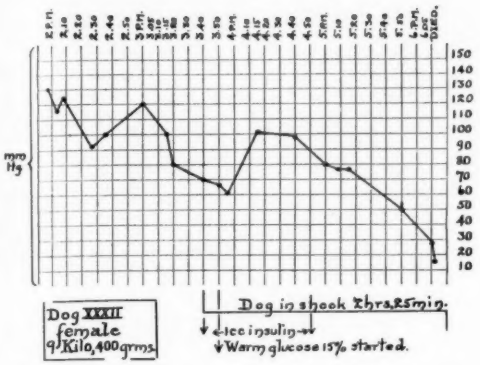

Fig. 1. Chart showing the blood-pressure changes in experimental shock treated with glucose and insulin.

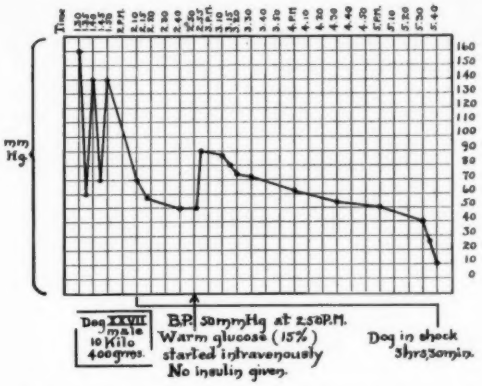

Fig. 2. Chart showing the blood-pressure changes in experimental shock treated with glucose only.

Fourteen of the dogs were treated with glucose and insulin (Lilly). The latter was used in a dosage of approximately 1 unit of insulin for every 2 grams of glucose given. The insulin was given subcutaneously before the introduction of the glucose in some animals and in others 20 to 30 minutes after the glucose was started. In a few experiments the insulin was given very slowly with the glucose by the intravenous method.

In all but one of the dogs, there was a rise in the blood pressure almost immediately following the administration of the hypertonic solution. The usual response was a rise to 80 or 90 millimeters of mercury. As a rule, this level was maintained from 15 to 30 minutes after which the pressure slowly fell and usually continued to fall steadily until the animal died from 1 to 4 hours after treatment was begun. In no case did an animal recover completely although one recovered a blood pressure of 100 and lived from 2:45 p. m. until midnight, or 9 hours and 15 minutes.

Six animals were treated with glucose alone to control the glucose-insulin treatment. The blood pressure readings in this series differed very little from those in the series receiving the insulin.

Shock was produced in the same manner in 12 dogs used as controls and progress was noted until death. The rapid fall in blood pressure with a continued low pressure until the end of the experiments corresponded very closely to the changes in blood pressure of the animals treated. Death occurred from ½ to 1 hour earlier than in the treated series. The transitory rise in pressure after the glucose was given, of course, was absent in the controls.

Finally, as further controls, 2 dogs were treated with 15 per cent glucose-insulin and 15 per cent sodium chloride solution. In one animal the glucose-insulin was given until the blood pressure fell to a critical level at which time the sodium chloride solution was substituted. In the other animal the procedure was reversed. The change from one crystalloid solution to another gave a slight temporary rise in blood pressure for a few minutes (a rise of about 10 millimeters of mercury) after which time the manometer reading fell as before the change of solutions.

In no instance was any evidence noted that the addition of insulin in any way caused any specific aid that could be determined by change in blood pressure or general condition of the animal. Glucose alone in 6 animals acted as favorably as the glucose-insulin combination. The hypertonic sodium chloride solution temporarily elevated the blood pressure similar to the glucose or glucose-insulin solution.

Several times during the production of the shock state, the blood pressure would spontaneously recover after being below 70 millimeters of mercury from 10 to 20 minutes. Thus, it is to be noted that any treatment

instituted before the tendency toward a falling pressure was definitely established, might appear to be of value.

In two dogs intravenous 15 per cent glucose given at the rate of 5 cubic centimeters per minute apparently caused a change in the hæmoglobin. The blood withdrawn after death in these cases was chocolate colored. This seemed to us sufficient evidence to warn against giving very rapidly glucose solution in this strength.

SUMMARY

1. In the interpretation of shock treatment, it must be remembered that mild cases often recover either with or without the use of the common intravenous solutions. Before an estimate is made as to the value of any special method, it must be definitely determined that the condition dealt with is really shock.

2. Hypertonic solutions of crystalloids have not been used with complete success in the past in the treatment of well developed shock. Recently, evidence is accumulating that intravenous therapy with hypertonic solutions of crystalloids, unless introduced with the greatest care, may be actually harmful.

3. From the experimental standpoint the glucose-insulin treatment of shock recently advocated by Fisher seems to be no more beneficial than treatment with a hypertonic solution of glucose or sodium chloride.

4. Further studies should be made to learn, if possible, whether a patient in the true shock state can utilize glucose with insulin as do normal individuals or patients suffering with other diseases.

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DEFENSIVE FACTORS IN CARCINOMA OF THE BREAST[1]

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UNTIL recently it has been practically impossible to give even a fairly accurate prognosis in any form of malignant disease. A consideration of the defense reaction against carcinoma led MacCarty, in 1910, to observe the presence of lymphocytic infiltration, fibrosis, and hyalinization around carcinoma cells in the breast and also the differentiation of these cells. He later applied the presence or absence of these factors in prognosticating the postoperative length of life. On this basis, Broders, in 1919, made a fairly accurate prognosis possible in cases of epithelioma, his prognostic data being based on a system of grading the degree of malignancy of these growths according to the relative degree of differentiation present.

LITERATURE

Most of the literature on the subject of defensive factors in carcinoma of the breast is the result of the work of MacCarty and Broders. In 1922, Sistrunk and MacCarty published a paper based on the study of 218 breasts which had been radically removed for carcinoma. The study was both pathological and clinical. They found that the greatest combined factors in longevity were cellular differentiation and hyalinization and that cellular differentiation, hyalinization, and fibrosis are the greatest single factors.

Again in 1922, MacCarty in a further study stated that when any of the factors are present, postoperative life is increased and increased most when differentiation and hyalinization are present. He stated that the uniform consistency of increased length of postoperative life in the presence of the defensive factors suggests that they play a significant rôle as a part of the natural defensive mechanism against carcinoma.

Later in 1922, MacCarty studied 92 cases of carcinoma of the breast in which death had occurred. He found that in the presence of fibrosis, postoperative life was increased 34 per cent and 40 per cent in the presence of hyalinization, and that when both were present, postoperative life was increased 56 per cent.

In 1924, in studying the prognostic value of the four factors, he found that whenever the factors are present the average length of life is greater than when they are absent, and that this is true of all four factors. He also found that the variation in the frequency of the presence of the factors in different organs suggests that the defense mechanism probably acts differently in different organs.

MATERIAL AND METHOD

This paper is based on the study of 300 breasts which had been radically removed because of carcinoma. For various reasons, only 222 were suitable for inclusion in the statistics. I examined every gross specimen and cut blocks from carcinomatous areas for microscopic study. All of the data have been checked by questionnaires. The living patients have been alive from 5 to 10 years without any evidence of recurrence. In all of the cases studied in which the patient died, death occurred within 5 years of the operation, and in every case it was due to recurrence or metastasis. The cases were divided into the following groups: (1) those cases in which the axillary lymph nodes were involved, the patient being alive after 5 years with no evidence of recurrence or metastasis, and (2) those in which the lymph nodes were not involved but death occurred within 5 years. These two groups were studied comparatively in order to determine the relative frequency of the factors in these two extremes. For comparison, the following groups were also studied: (1) those cases in which the lymph nodes were involved, but death occurred within 5 years, and (2) those in which there was no involvement of lymph nodes and the patient is living without evidence of recurrence or metastasis.

Throughout this paper, all references to involvement of lymph nodes refer to findings

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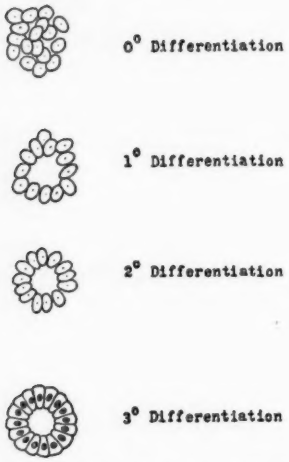

Fig. 1. Degrees of cellular differentiation based on the stages of differentiation of specific tissues during embryological development. In the stage of no differentiation, the cells are merely arranged in masses. In the first degree of differentiation, the cells assume the general alignment of the adult tissue without the arrangement of the axes of the cells as in the adult tissue cells. In the second degree of differentiation, the axes are parallel or form radii of a sphere or circles. In the third degree, the cells assume the adult morphological arrangement of the units of the specific tissue.

at the time of operation. When the number of years is stated, it indicates the interval since operation.

It is well known that in many cases of carcinoma of the breast in which lymph nodes were found involved at operation, the patient lives many years after without any sign of recurrence or metastasis. It is equally well known that in many cases in which only a small localized carcinoma is found, without involvement of lymph nodes or evidence of metastasis, the patient dies within a few months. It has been my aim in this study to find out, if possible, why this should be true. A clue to the answer may be discovered in MacCarty's work. If it is true that there is a definite defense mechanism, then it should further be true that this defense mechanism should be well developed in those patients who recover in spite of involvement of lymph nodes and that, on the other hand, cases of fatal carcinoma in which the lymph nodes were free should be characterized by a poorly developed defense mechanism.

The defensive factors dealt with in this study are the same as those observed by MacCarty, namely, differentiation, fibrosis, hyalinization, and lymphocytic infiltration, and the same definitions for the terms are used.

There is some doubt as to the correct position which cellular differentiation should occupy. It may be an inherent characteristic of the growth itself, or it may be due to factors in the surrounding tissues which allow differentiation. At first thought, cellular differentiation might naturally be considered as a characteristic of the growth and not connected with nature's defense, but this is apparently not the case. Differentiation of cells in nature is the result of adaptation to favorable environment. It is possible that this is also the case with malignant cells. It is thought that this favorable environment is the property of the organism rather than of the cell itself, and for this reason differentiation is placed among the body's defense factors. However, it is considered to be an index of the defensive mechanism rather than a specific defensive factor.

The microscopic sections were divided into three groups: those in which differentiation was absent or minimal; those in which the tendency to differentiate was distinct but not marked; and those in which the tendency to differentiate was marked (Fig. 1). Before the results were consolidated Groups 2 and 3 were combined so that the classification was based merely on the presence or absence of cellular differentiation. It is felt that this must play a very definite part and that a growth which is well differentiated should support a better prognosis than one which is not.

There was a distinct tendency to differentiate in about 65 per cent of all cases. The higher the degree of differentiation the more the growth resembled normal gland. In highly differentiated growths, it was quite commonly found that none of the other defense factors was present (Figs. 2 and 3).

In the study of fibrosis, the main difficulty was in determining in just what case fibrosis should be called a factor of defense and when it should not. If the presence of fibrous

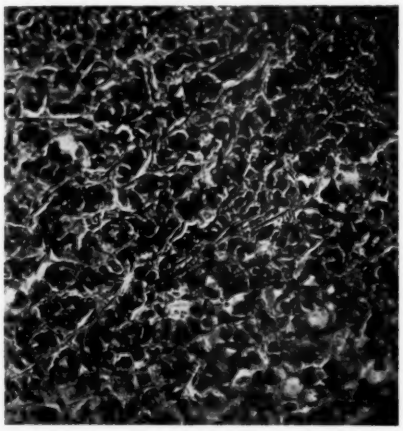

Fig. 2. An entirely undifferentiated growth with no defensive factors present.

tissue *per se* were to be taken as a positive criterion, fibrosis would be present in every case, since there is a certain variable amount of fibrous tissue present in every case. In the normal fibrous tissue, the fibrils present a characteristic interlacing pattern. Fibrosis due to malignancy, however, is in much more intimate relation with the cells. The fibrils form a barrier around the masses of cells which is quite different from the arrangement in normal fibrosis. The same question of degree was met with here and dealt with just as in the case of differentiation (Fig. 4).

The same statements made in regard to fibrosis are applicable to hyalinization, since hyalin develops from fibrous tissue. When hyalinization is marked, there is little pure fibrosis since the former is merely hyalinized fibrous tissue. Of all the factors, hyalinization is found in the smallest percentage of cases, probably because it is the last factor to appear. It was present in only about 30 per cent of the cases. Because of the firmness which characterizes it, hyalinization is the most effective of the tissue factors, since it constitutes a practically impossible barrier to the advance of malignant cells.

Lymphocytic infiltration is the least important factor; however, it is the first to appear. It is characterized by a dense wall of small lymphocytes, intimately surrounding

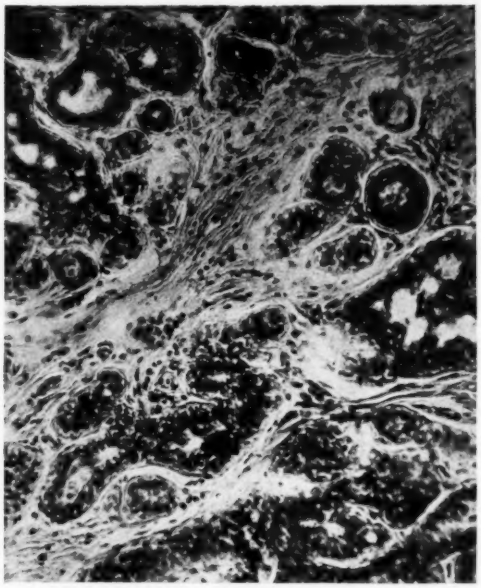

Fig. 3. Highly differentiated tumors. The cells are of a much more mature type than those in Figure 2. Nucleoli are not prominent and cells are placed in definite acinar groups. Here also there is very little defense reaction.

the cancer cells, which may be many cells in thickness (Fig. 5).

It was frequently found that when the degree of cellular differentiation was very high, there was little if any other defensive reaction. Frequently one factor, such as fibrosis or hyalinization, was also present to a marked degree while no other factors were present. The degree to which any factor is present undoubtedly plays an important part but as it was difficult to evaluate it, it is not included in the statistics.

DATA

In 88 cases, the lymph nodes were involved and the patient lived more than 5 years without recurrence or metastasis. In 20 of these cases, (23 per cent) all factors were present; in 87 (98.8 per cent) one or more, usually more, factors were present. In only 1 case were all of the factors absent. In this group as a whole, considering four factors for every case as 100 per cent, the percentage of factors present was 70 and the percentage of

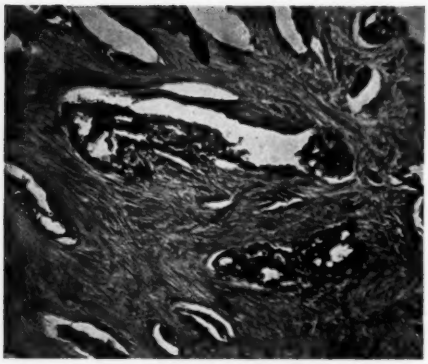

Fig. 4. Tumor in which fibrosis is very marked. Practically every group of cells has a dense fibrous ring around it, and shrinkage and degeneration of the malignant cells are quite pronounced.

factors absent was 30. This gives an average of about three of the four factors present in each case.

In 33 cases the glands were free from involvement but the patient died (Group 2). In 11 cases (33 per cent) none of the factors was present. In only one case were all four factors present. Taking this group as a whole, 73.5 per cent of the factors were absent, and only 26.5 per cent were present. This was an average of only one factor for each case, whereas in the former group, there was an average of three (Table I) as exemplified in such tumors as that illustrated in Figure 6.

Early in this paper, it was stated that if the so-called defensive factors were of any value, it would be shown in these groups. It was anticipated that in cases such as those in Group 1 the factors should be well defined, and that in such cases as those in Group 2 the factors should be poorly defined. Analysis of Table I shows that this is very definitely the case.

Comparison of the total incidence of the factors in the two groups shows that the defense mechanism was almost three times as manifest in Group 1. In Group 1, all four factors were present in 23 per cent or about one-fourth of the cases, while in Group 2, 3 per cent of the cases or only 1 case in the 33 had all four factors present.

In Group 1, only in 1 per cent of the cases were there no factors present while in Group 2 all factors were absent in 33 per cent. In Group 1 there were defensive factors in practically every case while in Group 2 there was absolutely no defense reaction in one-third of the cases.

TABLE I.—COMPARISON OF GROUPS 1 AND 2

	Group 1 Lymph nodes involved; patient alive (88 cases) per cent	Group 2 Lymph nodes free; patient dead (33 cases) per cent
Cellular differentiation present	86.5	33.3
Cellular differentiation absent	13.5	66.7
Fibrosis present	86.5	36
Fibrosis absent	13.5	64
Hyalinization present	55	9
Hyalinization absent	45	91
Lymphocytic infiltration present	52.5	27
Lymphocytic infiltration absent	47.5	73
All factors present	23	3
All factors absent	1.1	33
Total per cent of factors present	70	26.5
Total per cent of factors absent	30	73.5

On further analysis of Table I, it will be seen that cellular differentiation was about three times as common in Group 1 as in Group 2 being present in the former in 86.5 per cent of the cases and in the latter in only 33 per cent Fibrosis was present in about the same ratio, 86.5 per cent in Group 1, and 36 per cent in Group 2. The preponderance of hyalinization in Group 1 is even more marked; it was present in 55 per cent of the cases in Group 1, but in only 9 in Group 2. Lymphocytic infiltration was twice as common in Group 1—52.5 to 27 per cent. Not only were the factors as a whole much more common in Group 1, but each individual factor was much more common in Group 1 than in Group 2.

Fifty cases were studied in which the lymph nodes were free and the patients were still alive (Group 3). This group is not very distinctive as, in view of the early removal of the carcinoma, the prognosis should be fairly good, irrespective of the presence or absence of the factors. In 6 cases (12 per cent) all the factors were present. In 3 cases (6 per cent) all were absent. In the entire group, 61.5 per cent of the factors were present and 38.5 per cent were absent.

Fifty-one cases were studied in which the lymph nodes were involved and the patients were dead (Group 4). This group also is not very distinctive as the involvement of lymph nodes with or without the defensive

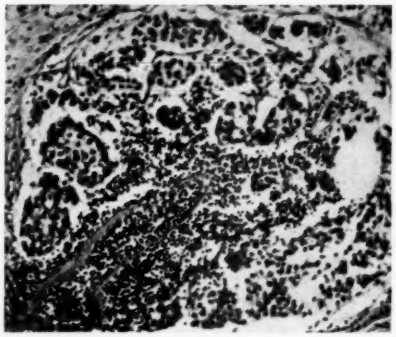

Fig. 5. Showing the lymphocytic infiltration around the cancer cells.

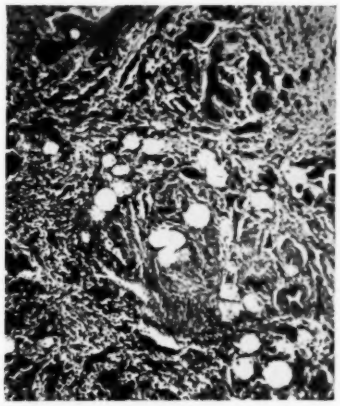

Fig. 6. Three factors are present: hyalinization and fibrosis are shown in upper half of the field and lymphocytic infiltration in lower left.

factors generally entailed a poor prognosis. Of this group, in 24 per cent, no defense factors were present and in 4 per cent all four factors were found. In this group as a whole, 61.3 per cent of factors were absent and 38.7 per cent were present. Even here, there was a marked preponderance of missing factors.

SUMMARY

Of the 300 specimens of carcinoma of the breast which were studied, for various reasons, only 222 were suitable for inclusion in the statistics. The study was confined to the four defensive factors, cellular differentiation, fibrosis, hyalinization, and lymphocytic infiltration. The study was undertaken with the idea of proving or disproving their defensive nature. They were found to play a distinct part in retarding the growth and spread of carcinoma. The various factors are discussed individually and observations on their characteristics are made.

In the 88 cases in Group 1 (the cases in which lymph nodes were involved but the patient was living more than 5 years after operation), 70 per cent of the defensive factors (an average of approximately three of four factors) were present. In 23 per cent of the cases, all factors were present. In the 33 cases in Group 2 (the cases in which the lymph nodes were free but the patient had died within 5 years), only 26 per cent of the factors were present, an average of only one of the four in each case. In 33 per cent of the cases, none of the factors was present, and in only 1 case were all the factors present. There was also a marked preponderance of the individual factors present in Group 1, most marked in the case of hyalinization, which was present in 55 per cent of the cases in Group 1 and in only 9 per cent of the cases in Group 2.

In the 50 cases in Group 3 (lymph nodes free and patient alive), factors were present in 61.5 per cent of the cases and absent in 38.5 per cent.

In the 51 cases in Group 4 (lymph nodes involved and patient dead), factors were absent in 61.3 per cent of the cases and present in 38.7 per cent, just reversing the figures of the previous group.

TABLE II.—COMPARISON OF GROUPS 3 AND 4

	Group 3 Lymph nodes free; patient alive per cent	Group 4 Lymph nodes involved; patient dead per cent
Cellular differentiation present	84	51
Cellular differentiation absent	16	49
Fibrosis present	80	49
Fibrosis absent	20	51
Hyalinization present	46	17
Hyalinization absent	54	83
Lymphocytic infiltration present	36	36
Lymphocytic infiltration absent	64	64
All factors present	12	4
All factors absent	6	24
Total per cent of factors present	61.5	38.7
Total per cent of factors absent	38.5	61.3

CONCLUSIONS

1. The preponderance of evidence indicates that cellular differentiation, fibrosis, hyalinization, and lymphocytic infiltration, are nature's defensive mechanism against carcinoma.

2. While these no doubt are not the only means of defense, they are probably the most important.

3. They play a definite and distinct part in retarding the growth of carcinoma in the breast.

4. They are not present in every case and vary considerably in degree and thus in effectiveness.

5. If, following amputation in a case without involvement of lymph nodes, all the defensive factors are found present, a good prognosis is justified; if they are not present, the prognosis is not so favorable.

6. If, in a case with involvement of lymph nodes, there are no factors present, the prognosis is poor. If all the factors are present, it is still not good, but certainly better.

7. The results emphasize more than ever the necessity of early diagnosis and radical amputation for carcinoma of the breast.

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GALL-BLADDER DISEASE IN YOUNG SUBJECTS

By ALFRED H. POTTER, M.D., Springfield, Ohio

THE incidence of gall-bladder disease in adolescents and patients under middle age has always been considered as very rare by most medical writers. Moreover, a perusal of standard works on pediatrics, including the latest and best textbooks written in the most interesting and presumably the most instructive way, would give one a wrong impression upon this subject; and would rather suggest that they cannot be based upon actual experiences outside of that of the authors since they hardly mention anything in reference to gall-bladder disease in children or at least imply that gall-bladder disease is a matter of but slight importance in childhood and adolescence. The writer's personal experience and study of the literature has, however, led him to a quite different opinion; and it is the purpose of this paper to show, with the help of collected data, that gall-bladder infections occur most frequently in adults under middle age, and that such diseases, including the occurrence of biliary calculi, may be met quite often in adolescents and young children.

The writer has within the past 3½ years personally observed 4 verified cases of gall-bladder disease in patients not over the age of 15 years, the histories of which are given at the end of this report. This fact was so unusual, and so much at variance with the accepted ideas on the subject, that the writer was led to investigate the literature of the age incidence of gall-bladder disease with special reference to infants, children, and young adults. This investigation appears clearly to establish the statement already made that gall-bladder disease is much more prevalent in young subjects than has hitherto been commonly believed; and furthermore that as regards the age incidence in diseases of the gall bladder and biliary passages, a restatement of our more exact knowledge and a correction of the misstatements upon which our beliefs in the past were based would appear to be absolutely essential.

To prove my contention that gall-bladder affections are found in patients under middle age of life more frequently than believed in the past, I will quote as follows: In a series of 259 operations on the gall bladder it was found that in over 15 per cent of the cases the patient was under 30 years of age.

Mann and Wilson (77), in a study of 57 cases of chronic gall-bladder disease in young adults operated on in the early stages of the infection found that more than 90 per cent were under 35 years of age.

Starr also states that in over 36 per cent of cases under observation, there was a history of biliary attacks before the age of 25 years.

STATISTICAL DATA

A study of statistics of something like 25,000 cases of gall-bladder disease, collected from the reports of well-known clinics and hospitals, shows the average age of patients at time of operation to be for females 36 years and for males 39 years. Although the duration of symptoms varies greatly, the study further shows that for females the symptoms last from the time of onset to the time of operation for an average of 6 years and for males, 5 years.

The shortest and longest duration from the time of onset of symptoms to the time of operation were 3 hours and 39 years, respectively. Of course, many factors intervene in regard to the time when patients seek surgical relief from their suffering. But the figures would apparently prove that the age of incidence of gall-bladder disease is on the average 30 years in the case of females and 34 years in the case of males. This is to say that, judged from the statistics, one must believe that gall-bladder infections begin to give symptoms in the most active period of life and then place the average age incidence far below that generally accepted in the past.

With regard to the occurrence of gall-bladder disease in very young subjects beginning with the case published by Gibson (58)

in 1722, and including cases reported up to the end of August, 1927, I have been able to collect 226 cases in this period in children. This number would, I am sure, be greatly increased if all observed cases had been reported. The review shows that pathological conditions of the gall bladder or ducts were observed in children under 15 years of age and were diagnosed by the symptoms and verified either by operation or autopsy.

An outstanding feature in this review is that the colored race is far less frequently affected with gall-bladder disease than the white race. Another matter, as regards the symptoms, is that while in many cases the symptoms in children were typical as in adults, yet children are rather inclined to complain of abdominal pain when gall-bladder disease is present, because they are apt to refer most pain to the abdomen. Indeed in adults many symptoms of cholelithiasis which are classified vaguely, as stomach and liver troubles, biliousness, indigestion, dyspepsia, etc., will be found in such a history, and will often lead to the diagnosis of biliary calculi before marked symptoms have presented themselves. Riedel (110) claims that "cramps" in the stomach are caused by gall stones in 97 out of 100 cases. When the so-called classical symptoms are present there is little difficulty in making the diagnosis.

THE OCCURRENCE OF GALL STONES IN YOUNG SUBJECTS

It is still believed by many that gall stones practically never occur during the first decade of life, and but rarely in the second. It is also believed that they are infrequently seen prior to the thirtieth year and that afterward they become progressively more common with advancing years.

In more than 9,000 autopsies, gall stones were found in 1 case in every 30 by Naunyn (93) in persons past 60 years.

It is stated by Hektoen and Riesman (60) that biliary calculi are found in 25 per cent of all cadavers at autopsy after the sixteenth year. It has been stated, however, that gall stones in patients under 35 years of age are quite unusual. With this statement, the writer does not agree.

Mathews (81) thought that at least one-half of the patients upon whom he had been called upon to operate for gall stones were under 30 years of age.

It is quite impossible from the collected statistics to determine definitely at just what age biliary calculi are formed, but review of the literature has shown that such calculi have been found at all ages from that of a 6-month fetus to a patient aged 86 years.

ETIOLOGICAL CONSIDERATIONS REGARDING GALL-BLADDER DISEASE IN YOUNG SUBJECTS

Certain etiological considerations would appear to support the occurrence of gall-bladder disease in young subjects.

In the past it was fairly accepted as a fact that typhoid fever was generally the preceding etiological factor in gall-bladder disease; but in recent years and at the present time it is accepted that other pathological conditions such as influenza, appendicitis, scarlet fever, diphtheria, etc., in the order named, can be preceding factors.

Medical literature shows a great decrease in the number of typhoid cases in the past few years; and if this decrease continues, typhoid will be practically eliminated.

The twenty-fourth annual report of the U. S. Census Bureau, shows a very great decrease in deaths from typhoid fever, even in the face of an estimated population increase. But this report also shows a great increase in deaths from influenza, and from this fact it is my belief that influenza is supplanting typhoid as the preceding etiological factor in gall-bladder disease.

The infectious diseases of children have, however, not received the attention they deserve as preceding factors in the evolution of gall-bladder disease; but I think that there can be but little doubt of the important rôle which these diseases play in infections of the gall bladder and biliary tract especially in the young.

Many conditions are a frequent concomitant, if not an etiological factor, and according to Jones and Joyce (65), in every case of pernicious anæmia they have come in contact with in the past few years, a chronic infection

of the gall bladder has been positively demonstrated. A small number of cases, which resembled mild pernicious anæmia, possessed the same type of biliary infection, and upon its removal the patients were restored to fairly normal health.

Digestive tract infections, no matter how they originate, must be considered the most probable origin of gall-bladder infections in the very young, but this presumes a stagnation of the gall bladder.

In cholecystitis, stagnation of the bile in the gall bladder is the main predisposing cause of infection. Hence the stagnant gall bladder may almost assume the importance of a clinical entity.

The gall bladder should not be viewed as an actively functioning organ that rhythmically fills and empties itself under the stimulus of digestion. An enormous concentration of bile in the gall bladder takes place with the addition of a mucous element. As stated by Mann (76) the most recent investigations have proved that the gall bladder has at least one function, viz., concentration of the bile, and plays a rôle in the regulation of intrabiliary duct pressure. But the flow of bile originates in the liver and sweeps down through the ducts into the duodenum disregarding in the main part the gall bladder; and variations in the bile flow and concentration occur in the liver in answer to the various degrees of food stimuli (7).

The gall bladder as an active organ does not seem to enter into the physiology of the bile excretion, the outflow from the gall bladder appearing to be in the nature of an overflow incontinence.

Because of the small amount of smooth muscle in the wall of the gall bladder, it is probably more correct to say the gall bladder evacuates rather than contracts.

Hydrops may be a condition affecting gall-bladder stagnation. As pointed out by Kehr (66), hydrops may be transitory or permanent. Acute infections of the gall bladder and cystic duct of a mild character usually are accompanied by hydrops. With the subsidence of the inflammation, the cystic duct recovers its function, drainage of the gall bladder occurs, and the hydrops disappears. Heisters' valves are swollen because of œdema of the mucous membrane of the cystic duct and this may be the cause of the already described condition. It is stated that a constriction of the sphincter of Oddi may be the cause of the same condition. But the most important factor controlling the overflow of bile from the gall bladder and biliary passages is the motility and tonus of the duodenum as pointed out by Burget (12).

I wish to report the 4 following cases of interest coming under my personal observation in the past 3½ years.

CASE 1. September 24, 1923. A well nourished boy, aged 11 years, had abdominal pain and vomiting for 5 days before examination. The only previous illness was measles, at the age of 6, influenza at the age of 10. The patient had had dizzy spells and vomiting several times during the past year. His skin was slightly jaundiced. He was drowsy without opiate. The right side of the abdomen was rigid and tender, with marked tenderness 2 inches to the right and upward from the umbilicus; tenderness over McBurney's point; distention and tympanic abdomen, especially in the epigastric region, very marked tenderness on palpation just below the twelfth rib, in the midaxillary line on the right side. The temperature was 102.4 degrees F.; pulse rate, 106; respiration, 44. The blood count showed: leucocytes, 23,400; polymorphonuclears, 94 per cent. Urinalysis was practically negative. A provisional diagnosis of acute appendicitis and acute cholecystitis was made. A laparotomy was performed that same night and showed a moderately inflamed, enlongated, high placed appendix, kinked and adherent at its mid-portion, and its distal end adherent in the region of the common duct. The latter was inflamed and thickened. Exploration of the gall bladder revealed a greatly enlarged, œdematous organ (hydrops), under great pressure. The wall was of a grayish color. It contained 178 cubic centimeters of mucus which was bile-streaked at the end of aspiration. The gall bladder was opened and explored; no stones were found. With some difficulty, water was forced through the ducts; the gall bladder was swabbed out with gauze and a rubber tube drain inserted. Pathological findings indicated that the condition was a subacute appendicitis. Culture of bile gave no growth. The patient had an uneventful recovery after appendectomy and cholecystostomy.

Postoperative notes: The discharge of mucus through the drainage tube stopped on the sixth day after operation; then bile escaped from that time on. The jaundice cleared up in 9 days after operation. The temperature was not normal until 10 days after operation. On March 17, 1927, 3½ years after the operation was performed, the patient was in excellent health.

CASE 2. A poorly developed boy, aged 6 years, presented a picture of severe illness. He had had but one previous illness of any consequence, viz., scarlet fever at the age of 4½ years and after which he had not enjoyed the best of health. His complaint dated from a few days after Christmas. He had attacks of colic-like pains in the abdomen; on several occasions he had diarrhœa, fever, anorexia, furred tongue, foul breath, and light yellow color of skin (not jaundiced). Examination, January 8, 1924, showed the face flushed; the skin light yellow in color; the chest negative; the abdomen greatly distended with some rigidity of the right rectus. He was very sensitive to pressure over McBurney's point and the epigastric region. There was seemingly no tenderness over the gall-bladder region. The temperature was 103 degrees F.; pulse, 120; respiration, 28. Blood count showed leucocytes, 13,000; polymorphonuclears, 78 per cent. Urinalysis was negative. A diagnosis of acute suppurative appendicitis was made.

At operation a right rectus incision was made over the appendix, and was carried rather high. The peritoneal cavity contained serous exudate. The appendix was rather highly placed; and the omentum completely surrounded it, but not firmly attached. The adhesions were released and delivered out of abdomen and removed. The appendix showed acute inflammation. As we were not satisfied that this would cause all the symptoms, the incision was lengthened, and exploration of the gall bladder revealed a moderately distended organ, which could not be emptied under gentle pressure. Palpation revealed a small conical shaped mass in the neck of gall bladder; but it did not have the consistency of a stone. Aspiration failed to withdraw the contents through needle, but when the gall bladder was incised, black tarry-like bile was spooned out. The bile was thick and contained sand. Without difficulty the conical shaped mass was removed from the neck of the gall bladder. It had the consistency of wet sand. A drainage tube was inserted in the gall bladder, and another was inserted in the right kidney region. The patient's convalescence was slightly stormy compared to Cases 1 and 3, although he made a complete recovery. A diagnosis of cholelithiasis, cholecystitis, and acute suppurative appendicitis was made. Cholecystostomy and appendectomy were performed.

The pathological findings showed an acute suppurative appendicitis. The bile gave a culture of mixed infection.

On January 10, 1927, the patient was a robust, healthy individual, enjoying good health.

CASE 3. A well-developed, but obese girl, aged 15 years, had had the usual diseases of childhood and diphtheria, and influenza in 1923. She had not had scarlet fever nor typhoid. On February 5, 1924, the patient gave a history of typical gall-bladder disease. In July, 1923, she began having what was diagnosed as indigestion. These attacks were sudden in onset, and accompanied by vomiting and pain in the epigastrium, radiating to the back via the ribs and right shoulder. There was no history of jaundice of the skin until the present time, although the scleræ were occasionally icteroid in color. There was soreness on deep palpation in the gall-bladder region; but there was no rigidity, and there were no palpable masses. She gave birth to a child on November 2, 1923, and since then had had attacks of pain every week in the gall-bladder region but with no definite relation to meals; and no chills or sweats. Occasionally light pasty stools were passed. The last attack occurred 1 week before admission and required an opiate. Her temperature was 97.8 degrees F. Pulse was 92; respiration, 24. Blood pressure was 110–65. Blood count was: leucocytes, 9,200; red blood cells, 3,150,000; hæmoglobin, 70 per cent. The patient nursed her baby up to the time of her entrance to the hospital. Urinalysis on February 5, 1924, showed a specific gravity of 1024; a trace of sugar; negative for albumin; acetone, negative; bile, negative. On February 9, the specific gravity was 1032; and there was 0.25 per cent of sugar. A pre-operative diagnosis of cholelithiasis, chronic cholecystitis, and chronic appendicitis was made.

On February 10, an operation with a high rectus incision was performed. Adhesions of the gall bladder, liver, intestines, and peritoneum were released. The gall bladder was normal in size; bile was aspirated and was of normal consistency. The organ was opened and explored. Thirteen facetted stones were removed. One stone in the cystic duct was milked back into the gall bladder and removed. A second stone in the cystic duct near the ampullar end, was crushed and removed. The other ducts were clear. A total of 15 stones was removed. The largest stone was the size of the end of the little finger. A rubber-tube drain was inserted in the gall bladder. Exploration of the appendix showed it to be retrocæcally located, adherent, and in a state of subacute inflammation. It was removed. Pathological findings indicated a subacute appendix. Culture of the bile was negative. The patient had an uneventful convalescence and was discharged on February 24, cholecystostomy and appendectomy having been performed. On March 1, 1927, the patient reported that she had been in good health since the operation.

CASE 4. A fairly well developed girl, small for her age of 4½ years and under weight was admitted September 10, 1926. She had pain in the right side, lower and upper quadrants and rigidity of the right rectus. She complained that vomiting and fever had been present for the past 2 days. She had had stomach trouble for 2 years, following influenza and had had three previous attacks with symptoms similar to those of the present illness. Examination revealed an abdomen greatly distended; rigidity of right rectus; tenderness over entire abdomen, marked at a point 2 inches to right and upward from umbilicus; slight tenderness over McBurney's point;

GALL-BLADDER DISEASE IN YOUNG SUBJECTS

No.	Year	Author	Sex	Age	Chole-lithiasis	Chole-cystitis	Jaundice	Pathology	Symptoms	How Diagnosed. Also Type of Operation
1	1722	Gibson	Male	12 Yrs.	Yes	No	No	Ascites. Many stones in common duct. Gall bladder distended	Pain, vomiting, loss of strength and weight	Autopsy.
2	1752	Coe	Unknown	Under 12	Yes	No				Autopsy.
3	1752	Lieutand	Male	25 days	Yes	No	Yes	Enlarged liver. Several calculi in gall bladder and ducts	Colic. Intense jaundice	Autopsy.
4	1813	Portal	Unknown	Infant	Yes	No	Yes	Liver infiltrated with blood		Autopsy.
5	1813	Portal	Unknown	Infant	Yes	No	Yes	Liver infiltrated with blood		Autopsy.
6	1813	Portal	Female	15	Yes	No	Yes	Bile ducts filled with biliary calculi	Depression, indigestion, œdema of arms and legs	Autopsy.
7	1822	Beverhoyt	Male	13	Yes					Autopsy.
8	1822	Beverhoyt	Unknown	Under 13	Yes					Autopsy.
9	1829	Cruveilhier	Unknown	6 mos.	Yes				Tuberculosis	Autopsy.
10	1829	Cruveilhier	Unknown	Infant	Yes					Autopsy.
11	1830	Orfila	Male	14	Yes		?	Dark green calculus, weight 2 gm.		Autopsy.
12	1834	Lolatte	Male	15	Yes	Yes	Yes		Abdominal pain, tenderness, distention and rigidity, constipation	Symptoms (stone found in fæces).
13	1835	Husson	Unknown	8	No	Yes		Peritonitis, perforation of gall bladder	Typhoid fever	Autopsy.
14	1838	Valleix	Unknown	Infant	Yes					Autopsy.
15	1838	Valleix	Unknown	New born	Yes					Autopsy.
16	1838	Valleix	Unknown	New born	Yes					Autopsy.
17	1843	Bouisson	Unknown	New born	Yes 3 stones		Yes	Commencing obliteration of common duct. Thick black bile in gall bladder	Abdominal pain. Intense jaundice	Autopsy.
18	1852	Archambault	Unknown	Infant	Yes	Yes		Ulcerative cholecystitis. Perforation of gall bladder	Typhoid. Peritonitis	Autopsy.
19	1853	Barthez and Rilliet	Unknown	12		Yes		Ruptured gall bladder with large abscess about it	Typhoid	Autopsy.
20	1861	Trousseau	Female	9	Yes	Yes?			Typical gall-stone colic	Symptoms.
21	1861	Wolff	Male	10	Yes					Symptoms (stone found in fæces).
22	1861	Frerichs	Female	7	Yes		No	Lardaceous disease of liver		Autopsy.
23	1864	Royer	Unknown	5	Yes	No	No	Common duct dilated. Perforated appendix. Analysis of calculi	Peritonitis	Autopsy.
24	1864	Chedevergne	Unknown	15	No	Yes		Ulcerative cholecystitis	Typhoid	Autopsy.
25	1869	Mangin	Female	11	Yes	Yes	Yes		Intermittent fever with attacks of colic	Autopsy.
26	1870	Senac	Unknown	5 to 10		Yes				Symptoms.
27	1870	Senac	Unknown	5 to 10		Yes?			Typical gall-bladder colic	Symptoms.
28	1870	Senac	Unknown	5 to 10		Yes			Typical gall-bladder colic	Symptoms.
29	1877	Cuffer	Male	12 days	Yes	Yes	Yes	Gall bladder reduced to small duct. Cystic and common ducts obstructed by calculi	Hæmophilia	Autopsy.
30	1879	Anduard	Unknown	Under 4	Yes				Colic	Symptoms (stone found in fæces).
31	1880	Semon	Female	5	No	Yes			Colic	Autopsy (stone in fæces).
32	1880	Massie	Female	4	Yes				Gall-bladder symptoms as in adults	Symptoms (stone found in fæces).
33	1880	Massie	Male	8	No	Yes?	No		Characteristic hepatic colic	Symptoms.
34	1880	Beale	Unknown (fetus)	8 mos.	Yes	No	Yes	Impacted stone in common duct		Autopsy.
35	1882	Walker	Male	3 mos.	Yes 3 stones		Yes		Colic	Symptoms (stone in fæces).
36	1882	Steadman	Female	13	No	Yes		Ulcerative cholecystitis. Perforation of gall bladder. Typical lesions of typhoid in intestines	Typhoid. Peritonitis	Autopsy.

GALL-BLADDER DISEASE IN YOUNG SUBJECTS—*Continued*

No.	Year	Author	Sex	Age	Cholelithiasis	Cholecystitis	Jaundice	Pathology	Symptoms	How Diagnosed. Also Type of Operation
37	1882	Steadman	Female	6		Yes acute		3 perforations fundus of gall bladder	Typhoid. Peritonitis	Autopsy.
38	1884	Mercat	Male	10	No	Yes	No		Attacks of severe epigastric pain, vomiting, tenderness over gall bladder	Symptoms.
39	1884	Mercat	Male	11	No	?	Yes	Hypertrophic liver	Biliary colic. Fever	Symptoms.
40	1884	Mercat	Female	3	No	?	Yes	Hypertrophic liver	Abdominal pain	Symptoms.
41	1884	Mercat	Male	3	Yes		No	Appendicitis, calculi in the appendix recognized as biliary	Peritonitis	Autopsy.
42	1884	Ricard	Unknown	Under 4	Yes		No		Abdominal colic	Symptoms (stone in fæces).
43	1884	Bond	Male	10	Yes	Yes	No	Typhoid, meningitis, perforation of gall bladder. Empyema of gall bladder	Of typhoid and pneumonia	Autopsy.
44	1889	Serveniere	Unknown	Under 15	Yes		No	*Note*—Serveniere collected 26 cases of biliary calculi in children under the age of 15 years	Typical gall-bladder colic	Operation ? Autopsy ?
45	1891	Roth	Female	Infant	Yes			Calculi blocking common and cystic ducts		Autopsy.
46	1892	De Haem	Female	2	Yes	Yes		Stone in cystic duct. Hydrops of gall bladder		Autopsy.
47	1892	Naunyn	Female	14	Yes				Colic	Cholecystotomy.
48	1892	Treeves	Unknown	10	Yes	Yes		Abscess of back from which calculus was discharged. Recognized as biliary	Indigestion and vomiting	Symptoms.
49	1893	Chiari	Unknown	12	No	Yes		Bronchopneumonia. Empyema of gall bladder ruptured	General peritonitis, typhoid, and pneumonia	Autopsy.
50	1896	Alexieef	Unknown	5	No	Yes acute		Rupture of gall bladder. Cultures from gall bladder showed bacillus typhosus	Peritonitis, pain, abdomen distended, typhoid fever	Operation. Drainage of abdomen.
51	1896	Battle	Male	6 mos.			Yes	Abdominal drainage evacuated pure bile. Autopsy revealed torn common duct	Acute peritonitis	Operation. Drainage of abdomen. Autopsy 9 days after operation.
52	1897	Courvoisier	Unknown	Child		Yes		Hydrops due to intussusception of cystic duct		Operation ? Autopsy ?
53	1897	Russell	Male	8	Yes	Yes	Yes	Distended gall bladder. Full of colorless thick mucus. Hydrops. At autopsy greatly dilated common duct	Fever. Severe gall-bladder colic. Pain in upper right side of abdomen	Cholecystostomy. Several days after operation. Autopsy.
54	1897	Charron	Unknown	Child		Yes		Hydrops of gall bladder	Typhoid fever	Operation ? Autopsy ?
55	1898	Wendel	Unknown	11 days	Yes 90 stones					Autopsy.
56	1898	Martin	Male	9				Free bile stained fluid in abdominal cavity. Gall bladder empty. Injury to duct	Abdomen distended, pain in right side, temperature 101° F., pulse 100. Retention of urine, bloody stools. No vomiting	Operation. Abdominal drainage.
57	1898	Ashby	Female	7	No	No	Yes	Large cyst apparently connected to liver. Autopsy revealed thick walled cyst of common and cystic ducts	Progressive emaciation, abdomen distended, œdema of feet. After 2nd operation patient developed peritonitis and succumbed	1-Drainage of cyst. 2-Choledochostomy. Autopsy.
58	1898	Thompson	Male	20 days	Yes	No	Yes	Commencing cirrhosis of liver. Several stones in gall bladder	Abdominal pain, weakness, constipation and light stools	Autopsy.
59	1898	Thompson	Unknown	Infant	Yes		Yes			Autopsy.
60	1898	Thompson	Unknown	Infant	Yes		Yes	Commencing cirrhosis		Autopsy.
61	1898	Thompson	Unknown	17 days	Yes		Yes	Commencing cirrhosis		Autopsy.
62	1898	Thompson	Unknown	17 days	Yes		Yes	Commencing cirrhosis		Autopsy.
63	1898	Thompson	Unknown	17 days	Yes		Yes	Commencing cirrhosis		Autopsy.
64	1898	Thompson	Unknown	17 days	Yes		Yes	Commencing cirrhosis		Autopsy.
65	1898	Ross	Female	3 mos.			Yes	Obliteration of common duct. Cirrhosis of liver		Autopsy.
66	1899	Still	Female	9 mos.	Yes 14 stones		No	11 stones in gall bladder. 3 stones in common duct. Pleurisy, bronchopneumonia and nephritis	Purpura, vomiting, wasting, constipation, clay-colored stools	Autopsy.

GALL-BLADDER DISEASE IN YOUNG SUBJECTS—*Continued*

No	Year	Author	Sex	Age	Cholelithiasis	Cholecystitis	Jaundice	Pathology	Symptoms	How Diagnosed. Also Type of Operation
67	1899	Still	Female	8 mos.	Yes 3 stones		No	Tuberculous meningitis. Miliary tuberculosis	Cerebral symptoms	Autopsy.
68	1899	Still	Male	5 mos.	Yes		No	Bronchopneumonia. Calculi in gall bladder	Severe abdominal pain, vomiting, pale stools, palpable liver	Autopsy.
69	1899	Still	Male	10	No	Yes	Yes	Viscosity of bile	Vomiting, abdominal pain, enlarged and palpable gall bladder	Symptoms.
70	1899	Still	Unknown	Infant	Yes	No	Yes			Autopsy.
71	1899	Still	Unknown	Infant	Yes		Yes			Autopsy.
72	1899	Still	Unknown	Infant	Yes					Autopsy.
73	1899	Still	Unknown	Infant	Yes		Yes			Autopsy.
74	1899	Still	Unknown	Infant	Yes		Yes			Autopsy.
75	1899	Still	Unknown	Infant	Yes		Yes			Autopsy.
76	1899	Still	Unknown	Infant	Yes		Yes			Autopsy.
77	1899	Still	Unknown	New born	Yes					Autopsy.
78	1899	Still	Unknown	Infant	Yes					Autopsy.
79	1899	Still	Unknown	Infant	Yes					Autopsy.
80	1901	Ogilvie	Female	15		Yes	Yes	During first week of attack of typhoid, round worm passed in fæces stained with bile	Abdominal colic. Typhoid fever	Autopsy.
81	1901	Rolleston	Male	6 mos.		Yes	Yes	Enlarged liver and spleen. Bronchopneumonia. Gall bladder small, cystic duct atrophied, common duct cord-like. Urine bile stained	Anorexia, vomiting, fever, loss in weight, clay-colored stools	Autopsy.
82	1901	Mitchell	Male	11	No	Yes acute		Distended gall bladder, W. B. C. 10,000. Cultures from gall bladder showed bacillus typhosus	Severe abdominal pain. Abdominal tenderness. Rigidity of muscles, visible abdominal tumor	Cholecystostomy.
83	1905	Riedel	Male	9	No	Yes acute	Yes	Bacillus coli in urine		Cholecystostomy.
84	1905	Sowers	Female	12	Yes	Yes acute		Gall bladder contained pus but no bile	Typical of acute infection of gall bladder. Temp. 102° F. W.B.C. 33,000	Cholecystostomy.
85	1906	Dever, H. C.	Male	13	No	No	No	Rupture of gall bladder	Fever, vomiting, vomitus greenish, rapid pulse, abdomen distended. Pain in right hypochondrium	Cholecystostomy. Pelvic drainage.
86	1908	Schlesinger	Unknown	4 mos.	Yes		Yes		Abdominal pain	Autopsy.
87	1908	Friedjung	Unknown	4 mos.	Yes					Autopsy.
88	1908	Ashhurst	Male	12		Yes?		Perforation of gall bladder. Peritonitis associated with bile stained fluid	Typhoid fever, severe abdominal pain, peritonitis	Autopsy.
89	1908	Miller	Female	13	No	Yes acute		Cultures of pus from gall bladder showed bacillus typhosus	Sharp abdominal pain, tenderness over gall bladder, rigidity of right rectus muscle. Temp. 102°F.	Cholecystostomy.
90	1908	Friedlander	Male	5	Yes	Yes	No		Colic, fever, distended gall bladder palpable	Cholecystostomy.
91	1909	Mayo Clinic	Female	15	Yes	Yes	Yes	Stone in cystic duct. Gall bladder œdematous. Hydrops	Pain under right costal margin for 1½ years. Tenderness over right hypochondrium	Cholecystectomy.
92	1909	Riedel	Female	13	No	Yes	No	Glandular swelling of hepatoduodenal ligament	Colic	Cholecystostomy.
93	1909	Montenbruck	Male	5	No	Yes	No	Acute hydrops, 250 c.cm. Bile aspirated. Greatly distended gall bladder	Pain, tenderness, palpable tumor	Cholecystotomy.
94	1909	Cahoy	Female	13	Yes	Yes		Small gall bladder showing fibrosis		Cholecystotomy.
95	1909	Warton	Male	13	No	Yes	No	Empyema of gall bladder. Acute appendicitis	Pain, tenderness in right hypochondrium. Palpable gall bladder	Cholecystotomy. Appendectomy.

GALL-BLADDER DISEASE IN YOUNG SUBJECTS—*Continued*

No.	Year	Author	Sex	Age	Cholelithiasis	Cholecystitis	Jaundice	Pathology	Symptoms	How Diagnosed. Also Type of Operation
96	1909	Warton	Female	15	No	Yes		Empyema of gall bladder 3 weeks after operation for acute appendicitis	Typical	Cholecystostomy.
97	1909	Cole and Winthrop	Female	15	Yes 30 stones	Yes	No	Culture of bile from gall bladder sterile	Epigastric pain, typical gall-bladder colic. Temp. 103°F, pulse 140. Extreme pain on pressure over gall bladder	Cholecystostomy.
98	1909	Stolzner	Male	7½	Yes					Operation ? Autopsy ?
99	1909	Churchman	Male	9	No	Yes	Yes	Acute typhoidal cholecystitis. Gall bladder contained thick bile, normal. W.B.C. 21,000	Severe abdominal pain. Right hypochondrium, tense and distended, gall bladder palpable	Cholecystotomy.
100	1910	Gleeson	Unknown	Child	Yes		Yes	Stones passed in fæces recognized as biliary calculi	Biliary. Colic. Catarrhal jaundice	Symptoms.
101	1910	Prince	Female	14	No	Yes		Thick walled gall bladder. W.B.C. 21,000	Pain in abdomen, and gall-bladder region. Fever, abdomen tender, marked spasm upper right rectus	Cholecystotomy.
102	1910	Childs	Female	13	No	Yes	No		Colic	Operation ? Autopsy ?
103	1910	Neuman	Female	13	No	Yes			Abdominal pain	Cholecystostomy
104	1911	Brock	Male	8	No	Yes	Yes	Enormous hydrops of gall bladder. 500 c.cm. fluid aspirated. Acute appendicitis	Slight jaundice, large palpable mass in region above McBurney's point, fever, pain and tenderness in upper and lower right quadrants, rigidity of right rectus	Cholecystotomy. Appendectomy.
105	1911	Ochsner	Female	10	No	Yes	No	Acute cholecystitis	Pain in abdomen, upper right quadrant	Cholecystostomy.
106	1911	Remsen	Male	15	No	Yes		Provisional diagnosis intestinal perforation. Appendix mildly inflamed. Gall bladder greatly distended. Cultures showed bacillus typhosus	Peritonitis	Cholecystostomy. Appendectomy.
107	1912	Marton	Unknown	2½	No	Yes	No	Provisional diagnosis intestinal obstruction. Greatly distended gall bladder	Abdominal pain and distention	Cholecystostomy.
108	1913	Khautz	Unknown	New born	Yes	No	Yes		Clinical observation	Symptoms. Autopsy ?
109	1913	Khautz	Male	5	Yes				Abdominal pain	Autopsy.
110	1913	Khautz	Male	7	Yes					Autopsy.
111	1913	Khautz		5	Yes					Autopsy.
112	1913	Khautz	Male	10	Yes	Yes				Autopsy.
113	1913	Khautz	Male	6	Yes					Autopsy.
114	1913	Khautz	Female	13	Yes					Operation.
115	1913	Khautz	Female	14	Yes					Operation.
116	1913	Khautz	Female	15	Yes	Yes			Abdominal	Operation.
117	1913	Khautz	Female	15	Yes	Yes				Operation.
118	1913	Khautz	Male	Under 15	Yes	Yes				Operation.
119	1913	Khautz	Male	Under 15	Yes	Yes				Operation.
120	1913	Khautz	Female	Under 15	Yes				Colic	Operation.
121	1913	Khautz	Male	12	Yes	Yes				Operation.
122	1913	Khautz	Male	Under 15	Yes					Operation.
123	1914	Pybus	Male	6	No	Yes acute		Pre-operative diagnosis sub-hepatic appendicitis with abscess	Pain in right upper quadrant associated with small swelling right hepatic region	Cholecystotomy.
124	1914	Else		New born			Yes?	Stricture of gall bladder		Autopsy.
125	1914	Else		New born			Yes	Stricture of gall bladder		Autopsy.
126	1914	Else		New born			Yes	Stricture of gall bladder		Autopsy.
127	1914	Else		New born			Yes	Stricture of gall bladder		Autopsy.
128	1914	Else		New born			Yes	Stricture of gall bladder		Autopsy.
129	1914	Else		New born				Stricture of gall bladder		Autopsy.

GALL-BLADDER DISEASE IN YOUNG SUBJECTS—*Continued*

No.	Year	Author	Sex	Age	Cholelithiasis	Cholecystitis	Jaundice	Pathology	Symptoms	How Diagnosed. Also Type of Operation
130	1914	Else		New born				Stricture of gall bladder		Autopsy.
131	1914	Eshner	Unknown	2	No		Yes	At autopsy—Liver normal size, duct except cystic normal, gall bladder absent, usual fissure for gall bladder wanting	Persistent cough, pale stools	Autopsy.
132	1914	Ayma Oms		Infant	Yes					Autopsy.
133	1914	Clark	Male	4 mos.	Yes	Yes		Congenital cardiac defect. Patent foramen ovale. Hydrops of gall bladder	Acute enterocolitis	Autopsy.
134	1914	Downes	Female	13	Yes 16 stones	Yes acute		Pre-operative diagnosis acute appendicitis	Of acute appendicitis	Cholecystostomy. Appendectomy.
135	1914	Paulsen		6 mos.	Yes					Autopsy.
136	1915	Babcock		4		Yes		Cholecystic toxæmia		Cholecystectomy.
137	1915	Babcock		14		Yes		Cholecystic toxæmia		Cholecystectomy.
138	1915	Buchanan		5½		Yes				Cholecystostomy.
139	1917	Eisendrath	Female	15	Yes 50 stones	Yes	No	Gall bladder distended, filled with purulent fluid. Cystic duct greatly distended with 4 stones	Pain in right hypochondrium radiated to right shoulder. Abdomen rigid, tender in right upper quadrant. Fever. Vomiting.	Cholecystectomy.
140	1917	Eisendrath	Female	15	Yes 1 cystic duct stone	Yes acute		Acute appendicitis. Acute hydrops	Severe pain right upper quadrant. Vomiting. Marked abdominal tenderness. Rigidity right iliac region	Cholecystectomy. Appendectomy.
141	1918	Smithies	Female	13	Yes	Yes				Cholecystostomy.
142	1918	Smithies	Male	15	Yes	Yes				Cholecystostomy.
143	1918	Albu				Yes		Cholangitis	*Note*—Albu describes a large number of cases which he designated enterogenous cholecystitis and cholangitis occurring in children during World War	Symptoms.
144	1918	Reid	Female	8	No	Yes		Gall bladder distended with 700 c.cm. of pus. Culture showed pure strain of bacillus typhosus. Cholecystitis with necrosis and hæmorrhagic	Fever, delirium, headache, diarrhœa, emaciation, anorexia, a high mass right side of abdomen. W.B.C. 10,400	Cholecystectomy.
145	1919	Chambers	Female	15	Yes		No	Pre-operative diagnosis cholelithiasis	Chilly sensations, fever, nausea, vomiting, acute abdominal pain, frequent attacks of symptoms	Cholecystectomy.
146	1919	Linder	Female	8	Yes	Yes acute		Gall bladder distended. W.B.C. 16,200	Abdominal cramps, tenderness right hypochondrium. Vomiting	Cholecystectomy. Appendectomy.
147	1919	Dever, H. C.	Female	5	No	Yes acute		Typhoid congested gall bladder with necrotic area in fundus. Purulent fluid from which hay bacillus was grown	Acute abdominal pain, peritoneal irritation	Cholecystectomy.
148	1919	Calloway	Male	14	Yes?	Yes		Typhoid	4 or 6 attacks of biliary colic in a year	Symptoms.
149	1920	Jacobson	Female	14	Yes many		Yes	Old pericholecystic adhesions and shrunken gall bladder	Abdominal tenderness, pain in upper right abdomen radiating to back and right shoulder. No nausea or vomiting	Cholecystectomy. Appendectomy.
150	1920	Jacobson	Male	13	No	Yes	Yes	Subacute cholecystitis. No stones	Pain and tenderness upper abdomen. Temp., 100.6° F. W.B.C. 8,000	Cholecystectomy.
151	1920	Kennedy	Female	13	Yes 4 stones			Appendicitis and intestinal worms	Abdominal pain, right hypochondrium rigid, vomiting, cystitis	Cholecystostomy. Appendectomy.
152	1920	Edington	Female	15	Yes 25 stones	Yes		Pre-operative diagnosis: Appendicitis. Wall of gall bladder nearly ¼ in. thick	Pyrexia, anorexia, vomiting, rigidity upper right rectus, tenderness over McBurney's	Cholecystostomy. Appendectomy.
153	1920	Bell	Female	14	Yes	Yes		Provisional diagnosis: Acute appendicitis and cholecystitis. W.B.C. 21,000. Gall bladder contained 2½ oz. of purulent pus	Fever, pain in lower abdomen in region of umbilicus, tenderness mid-abdomen and upper right quadrant	Cholecystostomy. Appendectomy.
154	1921	Freundlich	Unknown	Infant	Yes		?	Stone in common duct. Gall bladder normal		Autopsy.

GALL-BLADDER DISEASE IN YOUNG SUBJECTS—*Continued*

No.	Year	Author	Sex	Age	Cholelithiasis	Cholecystitis	Jaundice	Pathology	Symptoms	How Diagnosed. Also Type of Operation
155	1922	Howze	Unknown	5	No	Yes		Acute gangrenous cholecystitis	Fever, typical gall-bladder colic	Cholecystectomy.
156	1922	Drury	Female	9	Yes			Gall bladder full of pasty sand	Abdominal pain	Cholecystostomy.
157	1922	Drury	Female	9	No	Yes	Yes	Pre-operative diagnosis: Appendiceal abscess. Immensely distended gall bladder gangrenous	Severe abdominal pain, nausea, vomited continuously for 7 hours, anæmic, right rectus rigid, extreme tenderness over entire abdomen. Acutely ill	Cholecystectomy. Appendectomy.
158	1922	Farr	Male	13	No	Yes	Yes	Pre-operative diagnosis: Appendicitis	Fever, pain, rigidity, abdominal tenderness	Operation.
159	1922	Farr	Male	8	No	Yes	No	Appendicitis, scarlet fever	Fever, pain, rigidity upper right rectus, tenderness	Operation.
160	1922	Farr	Male	8	No	Yes		Abscess of liver	Fever, pain, vomiting, leucocytosis	Operation.
161	1922	Farr	Male	7		Yes	Yes	Ascites	Fever, chills, and vomiting	Operation.
162	1922	Farr	Male	12	Yes	Yes	No	Acute appendicitis	Pain, vomiting, tenderness, leucocytosis	Operation.
163	1922	Kellogg	Female	11	Yes 52 stones	Yes	No	Chronic appendicitis. Gall bladder œdematous and imbedded in dense adhesions	Fever, vomiting, rapid pulse, severe pain epigastrium and upper abdomen. Tenderness about navel and above and right costal arch. Rigidity right upper rectus	Cholecystectomy. Appendectomy.
164	1923	Potter	Male	11	No	Yes	Yes	Acute appendicitis. Acute hydrops. Gall bladder contained 178 c.cm. of mucus. Cultures negative	Fever, vomiting, right side rigid, distention epigastric region, tenderness over McBurney's and about navel and above. Leucocytosis	Cholecystostomy. Appendectomy.
165	1923	Pasley	Male	12	Yes large stone com. duct	Yes	Yes	Ascaris worm in biliary tract. Enlarged liver. Bile in urine	Fever, abdominal tenderness, rigid abdominal muscles, visible tumor, coated tongue, physical condition poor	Cholecystostomy. Choledochotomy.
166	1923	Joll	Female	14½	Yes many		Yes		Tenderness on deep pressure in right hypochondrium	Cholecystectomy.
167	1924	Reel	Female	12	Yes 1 stone	Yes	No		Fever, typical gallstone colic	Cholecystotomy. Appendectomy.
168	1924	Drury	Female	14	Yes many	Yes?	No	Gall bladder small, thick-walled, contracted containing stones. Stones in cystic duct. Gall bladder opened, stones removed. Appendix thick walled	Nausea, vomiting. No muscle rigidity, tenderness in lower right quadrant. No masses palpable	Cholecystectomy. Appendectomy.
169	1924	Lowenberg		Child		Yes	No	Acute suppurative cholecystitis with perforation and peritonitis	Typhoid fever, vomiting, abdomen rigid and distended	Operation ?
170	1924	Bittner		5		Yes		Acute suppurative cholecystitis with perforation and peritonitis	Typhoid fever, rigidity of abdominal muscles, vomiting, tenderness on pressure	Operation ?
171	1924	Bittner		9		Yes		Acute suppurative cholecystitis with perforation. Culture of pus from gall bladder showed bacillus typhosus	Typhoid fever, rigidity of abdomen, vomiting, tenderness	Operation.
172	1924	Bittner		11		Yes		Acute suppurative cholecystitis with perforation and peritonitis. Culture of gall-bladder pus showed bacillus typhosus	Typhoid fever, rigidity of abdomen, vomiting, tenderness, palpable gall bladder, pain on pressure	Operation.
173	1924	Potter	Male	6	Yes sand	Yes		Pre-operative diagnosis: Acute suppurative appendicitis. Culture of bile showed mixed infection	Pain in abdomen, diarrhœa, fever, anorexia, slight distended and rigid abdomen, tenderness over McBurney's but not over gall bladder. Leucocytosis.	Cholecystostomy. Appendectomy.
174	1924	Potter	Female	15	Yes 15 stones 2 in cystic duct	Yes	Yes	Pre-operative diagnosis: Subacute appendicitis, cholecystitis, cholelithiasis. Culture of bile negative	Fever slight, vomiting, pain in epigastrium radiated to right shoulder via ribs, tenderness on pressure over gall bladder and appendix	Cholecystostomy. Appendectomy.
175	1924	Fischer	Female	2½	No	Yes		Occlusion of common duct from compression of enlarged lymphatic glands	Scarlet fever	Cholecystotomy.
176	1924	Fischer	Female	9	No	Yes		Staphylococcus infection of gall bladder	Fever	Cholecystotomy.
177	1924	Fischer	Female	9 to 11	Yes					Operation ?

GALL-BLADDER DISEASE IN YOUNG SUBJECTS—*Continued*

No.	Year	Author	Sex	Age	Cholelithiasis	Cholecystitis	Jaundice	Pathology	Symptoms	How Diagnosed. Also Type of Operation
178	1924	Fischer	Female	9 to 11		Yes		Empyema of gall bladder	Typhoid fever	Autopsy ?
179	1925	Zeligs	Female (colored)	12		Yes acute		Bile aspirated through duodenal tube gave pure culture of bacillus typhosus. Positive Widal	Typhoid fever, pain in upper abdomen, spasm of abdominal muscles, rigidity, tenderness over entire abdomen, especially gall bladder region	Symptoms.
180	1925	Maxon	Male	4		Yes		Carcinoma of gall bladder		Autopsy.
181	1925	Erdman	Male	15		Yes		Carcinoma of gall bladder		Operation.
182	1925	Mayo Clinic	Female	13	Yes	Yes				Cholecystectomy.
183	1925	Mayo Clinic	Female	13	Yes					Cholecystectomy.
184	1925	Brickner	Female	1½	No	Yes	Yes	Enormous hydrops of gall-bladder congenital obstruction of biliary tract. Encephalitis. Palpable mass right liver border	Anorexia, diarrhœa, vomiting, fever, abdomen distended and rigid, positive Kernig, neck rigid, tetany of hands. Pulse 150, temp. 101° F, resp. 70, W. B. C. 17,500, polys. 64%	Cholecystostomy. Patient died several days after operation. Autopsy.
185	1925	Milch	Female	5		Yes		Pre-operative diagnosis: Acute appendicitis	Pain, fever, maximum tenderness of abdomen, well above umbilicus	Cholecystectomy. Appendectomy.
186	1925	Gehwolf	Female	8	Yes	Yes	Yes	Acute appendicitis. Stone in cystic duct. Cloudy exudate found in peritoneal cavity	Weak, slight jaundice, fever, abdomen distended, vomiting, diarrhœa, tenderness over McBurney's, pain in epigastric region	Cholecystectomy. Appendectomy.
187	1925	Roberts	Female	3	No	Yes staphylococcus	No	Hydrops of gall bladder, with acute inflammation. Gall bladder measured 1½ x 5 in.	Abdominal pain, constipation, vomiting, fever, rigidity of abdomen	Cholecystotomy.
188	1925	Patterson and Wyllie	Female	9	Yes 1 stone in cystic duct		No	Gall bladder thickened	Fever, abdominal pain, vomiting, tenderness right side of abdomen. No rigidity	Cholecystectomy. Appendectomy.
189	1925	Snyder	Male	4	Yes 1 stone	Yes	Yes	Pre-operative diagnosis: Acute appendicitis. Contents of gall bladder bile and pus. Culture showed streptococcus, staphylococcus, colon bacilli	Localized pain McBurney's, fever, nausea, vomiting, abdomen rigid and tender	Cholecystotomy. Appendectomy ?
190	1925	Snyder	Male	5½		Yes		Original diagnosis: Acute appendicitis. Gall bladder enlarged contained pus and thick bile, gave culture of mixed infection	Abdominal pain severe, nausea, fever, vomiting, W.B.C. 13,000, right side of abdomen rigid	Cholecystotomy. Appendectomy.
191	1925	Snyder	Male	9½		Yes		Diagnosis: Acute appendicitis cultures of bile gave mixed infection	Fever, pain, W.B.C. 16,000. Trace of albumin in urine	Cholecystotomy. Appendectomy.
192	1925	Singer		10	Yes	Yes				Operation ?
193	1925	Singer		10	Yes					Operation ?
194	1925	Singer		10	Yes					Operation ?
195	1925	Singer		10	Yes					Operation ?
196	1925	Singer		10	Yes					Operation ?
197	1926	Wronke	Unknown	8 days		Yes		Acute inflammation (hæmorrhagic diphtheritic) in bile ducts		Autopsy.
198	1926	Muller	Male	4	No	Yes		Gall bladder distended, thin-walled and greyish green. 50 c.cm. clear yellowish fluid withdrawn	Pain right side of abdomen, vomiting, constipation, pain in knees, tibias, and right arm; swelling to right of median line of abdomen from costal arch to umbilicus	Cholecystectomy.
199	1926	Key and Childe	Female	15	Yes 152 stones	Yes acute		Infected gall bladder	Acute abdomen	Cholecystectomy. Appendectomy.
200	1926	Dever, J. B.	Female	14	Yes				Typical	Operation.
201	1926	Judd	Male	8	Yes					Cholecystectomy.
202	1926	Potter	Female	4½		Yes	Yes slight	Pre-operative diagnosis: Acute suppurative appendicitis and cholecystitis. Peri-cholecystic adhesions. Elbow shaped gall bladder, thick-walled and dark color. *Postoperative* complication acetonuria	Abdomen greatly distended, rigidity of right rectus, tenderness over entire abdomen marked around umbilicus, fever, W.B.C. 20,000, polys. 86%. Patient acutely ill	Appendectomy. Peri-cholecystic adhesions released.

GALL-BLADDER DISEASE IN YOUNG SUBJECTS—*Continued*

No.	Year	Author	Sex	Age	Cholelithiasis	Cholecystitis	Jaundice	Pathology	Symptoms	How Diagnosed. Also Type of Operation
203	1926	Carey	Female	9	Yes stone in common duct	Yes		Appendicitis	Abdominal pain and rigidity, fever, vomiting	Cholecystectomy. Choledochotomy. Appendectomy.
204	1926	Bunch	Male	5	No	Yes		Pre-operative diagnosis: Intussusception. Empyema of gall bladder	Abdominal pain and rigidity, fever, vomiting, distention of abdomen	Cholecystostomy.
205	1926	Brown	Female	9	Yes stone in cystic duct	Yes		Pre-operative diagnosis: Acute appendicitis	Vomiting, tenderness over McBurney's point and gall bladder region. Right thigh flexed on abdomen	Cholecystotomy. Choledochostomy. Appendectomy.
206	1926	Clifton and Landry	Female	11	Yes	Yes	No	Pre-operative diagnosis: Acute appendicitis	Pyrexia, tenderness over McBurney's and gall bladder. Abdomen distended and rigid. Pain in right shoulder. W.B.C. 15,000, polys. 81%	Cholecystotomy. Appendectomy.
207	1926	Dever, H. C.	Female	12	No	Yes acute		Acute suppurative appendicitis. Bile thick and black	Sharp pains in abdomen, tenderness and rigidity lower right quadrant, clay stools, septic temperature	Cholecystostomy. Appendectomy. Drainage of abdomen.
208	1926	Crile	Female	8	No	Yes		Œdematous gall bladder		Operation.
209	1926	Dever, H. C.	Male	14	Yes 5 stones	Yes acute		Suppurative cholecystitis. Gall bladder distended	Shock; pain in upper right quadrant; board-like rigidity; vomiting	Cholecystectomy.
210	1926	Dever, H. C.	Male	13		Yes	Yes marked	Palpable mass size of orange in gall-bladder region	Fever, vomiting, loose yellow stools, restlessness, tenderness and muscle spasm in gall-bladder region	Cholecystostomy ?
211	1927	Quoted by Mitchell	Female	6 mo. fetus	Yes stone in common duct					Autopsy.
212	1927	Mayo Clinic		5 mos.	Yes					?
213	1927	Mayo Clinic	Female	11	Yes		Yes	Stone in common duct		Choledochotomy.
214	1927	Mayo Clinic		3	Yes					Cholecystectomy.
215	1927	Mayo Clinic		9	Yes					Cholecystectomy.
216	1927	Mayo Clinic		9	Yes					Cholecystectomy.
217	1927	Mayo Clinic		Under 15	Yes					Cholecystectomy.
218	1927	Mayo Clinic		15	Yes					Cholecystectomy.
219	1927	Mayo Clinic		Under 15	Yes					Cholecystectomy.
220	1927	Mayo Clinic		Under 15	Yes					Cholecystectomy.
221	1927	Mayo Clinic		Under 15	Yes					Cholecystectomy.
222	1927	Mayo Clinic		Under 15	Yes					Cholecystectomy.
223	1927	Mayo Clinic		15	Yes					Cholecystectomy.
224	1927	Mayo Clinic		15	Yes					Cholecystostomy.
225	1927	Mayo Clinic		Under 15	Yes		Yes	Hæmolytic jaundice		Cholecystectomy. Splenectomy.
226	1927	Mayo Clinic		15	Yes		Yes	Hæmolytic jaundice		Cholecystectomy. Splenectomy.

Added Cases:—Female, 4½ years, cholelithiasis; female, 8½ years, cholangitis.

skin of lemon color. Temperature 103 degrees F.; pulse, 136; respiration, 44. The blood count showed white blood cells 20,000; polymorphonuclears, 96 per cent. Urinalysis was negative. Preoperative diagnosis: acute suppurative appendicitis and acute cholecystitis. A laparotomy was immediately performed and revealed the appendix retrocæcally located, bound down, distended, covered by the meso-appendix and omentum; with gangrenous tip. It was removed. Palpation of the gall bladder revealed a moderately enlarged organ, which could not be emptied under gentle pressure. Very dense peri-cholecystic adhesions between the gall bladder and the duodenum (elbow shape gall bladder) were released. We could then empty the organ under gentle pressure. The walls of the gall

bladder were thickened and very dark in color. On account of the child's condition, the gall bladder was not drained; no stones could be palpated. Convalescence was retarded and complicated with acetonuria. Postoperative diagnosis: acute gangrenous appendicitis, acute cholecystitis. Pathological examination revealed a gangrenous appendix. Postoperative notes, September 11, 1926: Urinalysis revealed acetone and diacetic acid: three plus. This condition persisted until September 22. Temperature 103 to 104 degrees F.; pulse, from 120 to 140; respiration 44 to 48 up to September 21. Temperature receded from 101 degrees (September 22) to 99 degrees F. (September 27), when it became normal. Patient was discharged on the twentieth day. March 19, 1927, patient was enjoying good health.

STATISTICAL SUMMARY

Sex	
Males	62
Females	74
Not stated	90
Total	226
Ages	
Fetus (6th and 8th month, Cases 211 and 34)	2
Newborn	12
Very young (age in days)	9
Infant	19
Child	4
Less than 1 year (1 month to 1 year)	14
Between ages of 1 and 5 years	26
Between ages of 5 and 10 years	55
Between ages of 10 and 15 years	85
Total	226
Calculi	
Present	140
Absent	48
Not stated	38
Total	226
Jaundice	
Present	64
Absent	34
Not stated	128
Total	226
Cholecystitis	
With calculi	44
Without calculi	59
With jaundice	30
Not stated	93
Total	226
Calculi in ducts	
Cystic	14
Common	13
Hepatic	0
Not stated	199
Total	226
Hydrops of gall bladder	18
Infection of gall bladder	43
Malignancy of gall bladder (Cases 180 and 181)	2
Largest number of calculi found (Case 199)	152
Diagnosis	
By symptoms	21
Operations	126
Autopsy	79
Total	226

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BRAIN-FLAP—AN INFREQUENT OCCURRENCE IN CLINICAL SURGERY

Its Experimental Production

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In 1923 Sir William Wheeler (12) described a remarkable condition which he had observed during the course of an intracranial operation.

The patient had been admitted to hospital with his forehead bruised and cerebrospinal fluid trickling from his nose. He was breathing heavily, and when his skull was opened a marked rise and fall of the frontal lobe occurred with respiration. Another feature of the case was the presence of air beneath the dura, a condition which has been termed "pneumocephalus" or "intracranial aerocele."

With the exception of Wheeler's observation, we have found no reference to any mention of a marked respiratory excursion of the brain in the course of an operation, but it has since been the fortune of one of us (A. K. H.) to observe this condition on four occasions.

In the first case, the condition appeared toward the end of an operation for hydrocephalus in a child, performed by a former colleague, Mr. A. A. McConnell, at the Richmond Hospital, Dublin.

In the second, there was a leak of cerebrospinal fluid from a dilated cerebral ventricle which had been opened during an exploration for abscess of the temporal lobe.

In this case the expiratory excursion of the brain was first seen at the trephine opening on the tenth day after the operation while the case was being dressed, and it was then demonstrated to our colleagues, Prof. Biggam and Dr. Barrada.

In the third patient, who had lost much blood and was breathing heavily, very large cerebral movements were observed after the removal of an extensive depressed fragment of the frontal bone, just in front of the vertex, which had deeply indented the dura without actually piercing it. These large movements were observed for a period of 5 minutes during the closure of the wound.

The fourth patient had suffered an injury so similar to that in the third case, that, even before the skull was opened, we ventured to predict a respiratory movement of the brain. This occurred at once on the raising of the depressed fragment of the bone and synchronized exactly with a deep inspiratory gasp, but, after 3 or 4 diminishing excursions, the brain ceased to move. In this case, the bone fragment had penetrated into the (superior) sagittal sinus.

Dr. Ahmed Handousa, surgical registrar to Kasr el Aini Hospital, who operated on the fourth case, has since observed an exactly similar condition in a fifth case, the occurrence of the movement, which was large at first, which synchronized with respiration, and which proved to be only transient.

In every one of our clinical cases it was clearly seen—in the first 3 repeatedly—that, with expiration, the brain bulged outward toward the opening in the skull and that it receded inward from the opening with inspiration.

These unusual movements of the brain are so large that, from analogy with the flapping of the mediastinum which occurs with respiration in open pneumothorax, we have ventured to coin the name, "brain-flap."

In the first instance, we were led to investigate this phenomenon by experiment because we believed that the occasional presence of air within the cranial cavity after fracture of the skull might be found to be in some way connected with t.

It was soon found, however, that while the apparent explanation of "brain-flap" in terms of venous aspiration during the inspiratory suck of the thorax might at first sight

seem obvious and complete, the condition, in fact, afforded a problem of some complexity. For this reason, we have at present confined our experiments to its elucidation, leaving the original question of intracranial aerocele for further study.

LITERATURE

With the exception of the paper by Wheeler, already referred to, we have been unable to find any reference to the phenomenon of "brain-flap" in any surgical publication. It would seem that Wheeler himself was also unaware of the existence of any such publication.

The textbooks, both of surgery and physiology, available at the time, were also silent on the point. Again, such journals as were accessible gave no hints as to whether any work had been done on this subject or not. In consequence we decided to perform the experimental work and hoped that, during the summer vacation, one of us would be able, on his return to England, to make a further search into the literature. This was fortunately the case, and, through the kindness of Sir Charles Sherrington, we are able to cite some earlier work of which, previously, we had been in ignorance.

Mosso (7) was able to examine the movements of the brain in 3 cases in man, in which, through injury or disease, the coverings of the brain had been destroyed. He observed pulsations due to both the circulation and the respiration. To us it is somewhat surprising that his recorded changes in volume due to respiration are so small in comparison with those due to the circulation. In our work, as will appear later, the latter were only occasionally observed while the former might be, and often were, very extensive. Mosso attributed the changes in volume to variations in the blood content of the skull following on the respiratory movements. Naturally, he was unable to determine the effects produced by such procedures as section of the vagi.

A paper by Roy and Sherrington deals with variations in cerebral volume (8). The work there described was undertaken with the desire to determine the presence or absence of a vasomotor supply to the cerebral vessels. There were certain differences in technique between their experiments and ours; and, if we remember the differences between their objects and ours, it is not surprising that they only incidentally mention the effects of respiration on the cerebral volume. As far as the procedure in the two sets of experiments is similar, the results show a close resemblance, a fact which gives us the greater satisfaction, in that our work was done in complete ignorance of the earlier publication.

These two publications are the only papers in which we have been able to find accounts of work done under conditions similar to our own, in that the cranium has been opened and its contents exposed to atmospheric pressure during the whole of the experiment. Hill (3) has published the results of experiments on the actual cerebral volume but with the trephine opening blocked. All other work, such as that of Knoll (4), Becht (1), Weed (10), and very many others, deals solely with intracranial pressure, and almost always under the conditions of a closed cranium.

EXPERIMENTAL METHODS

For the purpose of this research, dogs, of from 4 to 15 kilograms in weight, have been used. In the earlier experiments, ether, given by the "open" method, was used as the anæsthetic, but it was soon found preferable to adopt the method of intratracheal insufflation of ether, using Palmer's "ideal" respiration pump.

Blood pressure. The systemic blood pressure was recorded by means of a mercury manometer connected to a cannula inserted into the femoral artery, usually on the right side. A cannula inserted into the femoral vein of the other side, permitted such intravenous injections as were required to be made.

Respiration. Various methods of recording the respiration were used, such as Gunn's stethograph (2). For reasons which will become apparent in the latter part of the paper, methods which involve the recording of movements of the thorax or abdomen were discarded in favor of one which enables variations in the intrathoracic negative pressure to be recorded. For this purpose, two glass tubes, one long and one short, were fitted by means of rubber corks to a two-necked *Woulfe's* bottle of a capacity of 1 liter and about three-fourths filled with water. To the long tube, which opened well below the surface of the water in the bottle, a short thin walled metal tube was connected by means of rubber tubing. The other extremity of the metal tube was fashioned into a beveled end. The short glass tube was similarly connected to a delicate tambour. A small incision was made through the skin and deep fascia over an intercostal space and the beveled end of the metal tube inserted. By means of a pursestring suture the

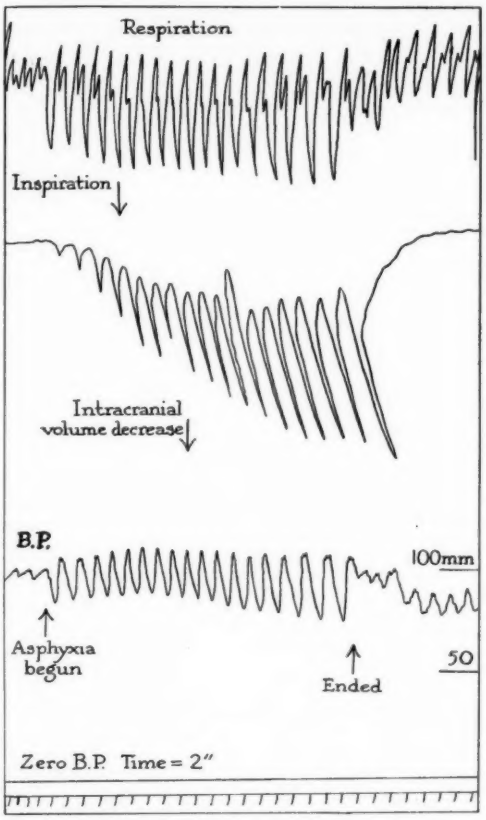

Fig. 1. The uppermost line represents the changes in negative pressure in the thorax, lowering of pressure i.e., inspiration, causing the lever to move downward. The middle section records changes in intracranial volume, reduction causing the lever to descend. The next part corresponds to the systemic blood pressure. Below this are two lines, the upper, giving the level of zero blood pressure, and the lower, intervals of time corresponding to 2 seconds. The figures at the right hand side correspond to the height at which the blood pressure recorder would stand for definite levels of pressure. "Asphyxia" is used as a convenient abbreviation for "occlusion of the tracheal cannula." These observations apply, *mutatis mutandis*, to all further tracings, and no further repetition of these remarks will be required. So far as possible, in this and all other tracings, the various lines have been so arranged that vertical lines drawn from top to bottom, would show synchronous changes in each line.

On commencement of "asphyxia," there are, immediately, large changes in intrathoracic pressure. Similarly, the intracranial volume lever, previously steady, shows rapid movements, growing more pronounced and at the same time there is an increasing lowering of the point to which it returns, increasing to a maximum. The blood pressure remains generally steady although the respiratory waves are greatly enlarged. At the end of "asphyxia," there is an almost complete cessation of the movements of the volume lever and the original level is rapidly reattained. The respiratory movements remain large on account of the previous deprivation of oxygen.

skin was drawn closely around the tube to form an airtight joint. The tube was then thrust through the intercostal muscles, so producing a connection between the pleural cavity and the Woulfe's bottle. Any variation in the pressure within this cavity will cause the water in the long tube to rise or fall, which in turn will abstract from or add to the water in the Woulfe's bottle and so vary the pressure in the air space above the water. The tambour will be affected by the variation in pressure and the lever will fall with a decrease and rise with an increase of intrathoracic pressure. Given a sufficiently large tambour it would be possible to connect the intrapleural cavity direct to the tambour, but the changes of pressure are so great that the size of tambour required would be quite impracticable. The Woulfe's bottle is inserted into the system to reduce the pressure variations to be recorded and the size of the tambour required. The larger the amount of water in the bottle, the larger will be the movements of the tambour lever for any given change of intrapleural pressure and vice versa.

Brain-flap. To obtain records of the variations of intracranial volume which form the subject of this paper, the temporal muscle on one side, usually the left, was reflected and a hole cut in the parietal eminence by means of a trephine, 2 centimeters in diameter. Care was taken to avoid damaging the dura mater, and twice only in the whole series of experiments did such injury occur. Sometimes there was severe hæmorrhage from the diploe, which was controlled by means of a piece of muscle and pressure. When bleeding had ceased, an instrument, made specially for this purpose, was inserted into the opening. This was made for us by Mr. Price, late of the Physical Department of the Egyptian Ministry of the Interior, to whom we desire to express our gratitude. A piece of brass, 3.5 centimeters in diameter, was turned down at one end to a diameter of 7 millimeters. The other end was fashioned into a cone of the exact dimensions of the hole left by the conical trephine. A flange, of the full diameter of the brass, was left at a distance of 7 millimeters from this end. The conical portion was bored out to an internal diameter of 16 millimeters and on the outer conical surface a fine but deep screw thread was turned. A hole 5 millimeters in diameter was then drilled from the narrow end to join that from the wide end. This instrument was screwed into the opening in the skull, and to insure an airtight joint, a strip of vaselined gauze was wound, between the skull and the flange, round the junction of brass and bone. Connection was then made by means of rubber tubing to a volume recorder, made by Messrs. Palmer, of such delicacy that a change of intracranial volume of one-tenth of a cubic centimeter caused a movement of the writing point on the surface of the drum of 2 centimeters of arc.

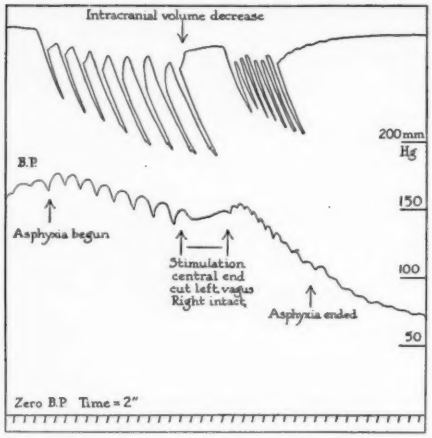

Fig. 2. To show the dependence of brain-flap on respiration. The condition being well established by "asphyxia," the central end of the cut left vagus nerve was faradized. Respiration ceased at once, as did brain-flap. Both commenced again at once on cessation of the stimulation. Although there is no respiratory tracing shown, it may be seen from the absence of respiratory waves on the blood pressure tracing during the period of stimulation that the respiration had, in fact, ceased.

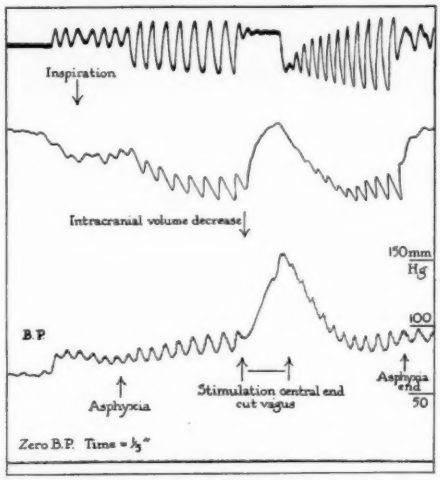

Fig. 3. This chart shows tracings which indicate essentially the same results as those shown in Figure 2. In this case, both vagi had been cut and consequently stimulation caused a large rise of blood pressure with an accompanying increase in intracranial volume, superposed on the cessation of movements due to the stoppage of respiration.

EXPERIMENTAL RESULTS

In the earlier stages of the work, no attempt was made to record the changes in intracranial volume, observation only of the dura mater being considered sufficient. The movements of the membrane were soon obtained and found to be quite extensive, as it was seen to fall in, on occasions, to a depth of several millimeters. It was soon found that the problem was by no means simple, several factors apparently entering into the production, or the reverse, of the movements. It was found that, in different animals under apparently the same experimental conditions, the required condition might or might not appear, and that, when present, the variations in intracranial volume might differ very considerably.

As soon as an experimental method had been found which could be expected to give successful results with reasonable constancy, the recording of these variations in intracranial volume was taken in hand and experiments performed with a view to elucidating the nature of the factors concerned and the share taken by each in the production of brain-flap. The experiments performed in connection with each of these factors will now be considered seriatim.

Influence of Respiration

In the early experiments it was soon recognized that the sinking in of the dura, if present, was practically synchronous with inspiration. It was also found that, with normal quiet respiration, such movements of the writing point on the drum as might occur were very small, in spite of the great magnification produced by the lever. If, on the other hand, there existed any impediment to the entry of air to the lungs, such as the presence of mucus in the trachea, these movements at once became larger. Attempts were then made to impede the air entry. This was done in the earlier experiments by wrapping a cloth around the animal's muzzle and forcibly compressing the mouth and nose with the hand. In the later work, when the method of insufflation of ether through a tube in the trachea had been adopted for anæsthetizing the animals, the openings of the cannula were closed with the finger and thumb, a far more efficient method.

It was then found that, though brain-flap, for reasons to be adduced later, was not necessarily induced by this procedure, the extent of the movements, if present, was very greatly enlarged or, if not previously present, they might be caused to occur. Again, the instant the obstruction to air entry was removed, the size of the movements immediately became lessened, rapidly returning to that which had obtained before the interference with respiration had been effected. This was the case in spite of the fact that the animal was often markedly hyperpnœic as a result of the deprivation of oxygen (Fig. 1).

In view of these facts, the method of recording variations in the negative pressure in the pleural cavity was adopted. It was thought that this would give a far clearer idea of the conditions obtaining in that cavity as a result of respiration than could be given by any method dependent on the measure-

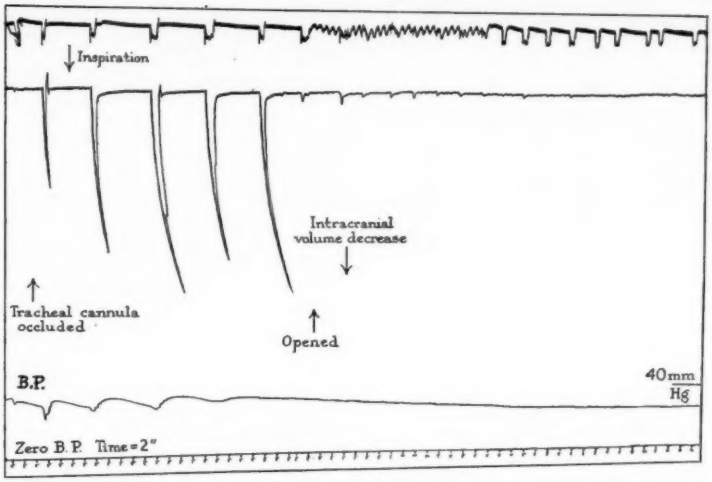

Fig. 4. A tracing from a moribund dog in which respiration took the form of gasps. The immediate appearance and disappearance of brain-flap on occlusion and release of the tracheal cannula are characteristic. The synchronicity of the movements of the volume and respiration levers is well marked.

ment of changes in diameter or volume of the chest or abdomen.

Division and stimulation of the vagus. Another method of demonstrating the close association between brain-flap and respiration was sought. If one vagus nerve be divided in the neck, the other being left intact, stimulation of the central end of the cut nerve will cause cessation of respiration with little or no accompanying change in the systemic blood pressure. Brain-flap was induced in an animal and, when the movements of the intracranial volume lever were pronounced, stimulation with the faradic current of the central end of the cut vagus was commenced. At once the movements of the lever recording volume and respiration ceased. Immediately the stimulus was stopped, they recommenced. This is displayed in the tracing reproduced as Figure 2. If both vagi are divided, stimulation of the central end of one causes a considerable rise of the systemic blood pressure, due to stimulation of the vasomotor center alone, without the concurrent depressor reflex which will be present if one nerve is left intact. In consequence the results obtained under these conditions are of less value, as will be more readily appreciated when the experiments described in the next section of the paper are considered (Fig. 3).

Gasping. Figure 4 is a reproduction of a tracing obtained from a moribund dog. In this animal respiration consisted only of a series of gasps. While the air entry was impeded, the movements of the levers recording variations in intracranial volume and respiration respectively will be seen to be exactly synchronous, both moving downward with inspiration and upward with expiration. When the obstruction to respiration was removed, there was a short period of more rapid respiration, soon followed by a recurrence of gasping. It will be observed that, though the movements of brain-flap are well marked while the tracheal cannula is blocked, they cease at once on the removal of the obstruction to air entry and do not recur with the reappearance of gasping.

Association of brain-flap and respiratory movements with inflated or deflated chest. Another type of experiment was performed to demonstrate the dependence of brain-flap on a further decrease of the normally negative intrathoracic pressure. Figure 5 is a reproduction of a tracing which may be divided into two parts. In the first, the tracheal cannula was occluded when the chest was at its smallest volume at the *commencement* of inspiration. When the cannula was closed, the pressure changes in the pleural cavity were immediately greatly increased, the negative pressure becoming more negative with inspiration. Brain-flap appeared at once, the movements of the lever being practically at their maximum from the beginning. In the second part, however, the air entry to the lungs was cut off with the chest distended at the *end* of inspiration. Under these conditions, a period of time elapsed before the changes in intrapleural pressure with respiration became much increased. At the same time, as can be seen from the tracing, brain-flap began at once but the movements were very much less marked, being very small at first and gradually increasing to about half the size of those obtained in the earlier part of the tracing.

Stimulation of the phrenic nerves. It was thought that information might be gained from the results of artificially induced movements of the diaphragm.

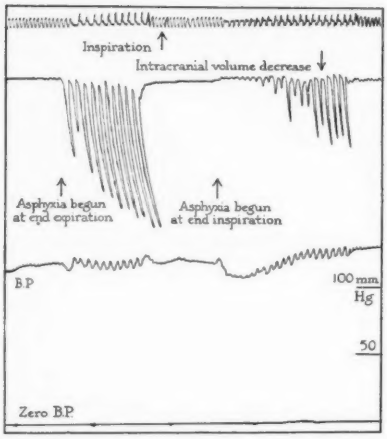

Fig. 5. To demonstrate the relationship between brain-flap and the degree of expansion of the thorax. In the first part asphyxia was commenced at the end of expiration. The movements of both the respiration and volume levers are nearly maximal to begin with. In the second part, asphyxia was begun at the end of inspiration. The movements of both levers are small to begin with, gradually growing larger but never attaining the size of those in the first part. The respiratory tracing was made with a stethograph in this experiment, the lever moving upward with inspiration.

The phrenic nerve was isolated in the neck on both sides. This is not easy in the dog as the nerve arises as branches of the brachial, not of the cervical, plexus. Because of this, it was usually impossible to isolate more than a part, perhaps two-thirds, of the nerve without the risk of injury to the pleura or great veins. The nerves were then stimulated simultaneously, with the electrodes placed in series so as to insure the receiving of the same amount of current by both nerves. Three types of stimulation were employed: single shocks, short and long faradizations. The tracheal cannula was occluded before, but only just before, the commencement of the stimulation.

Figure 6 shows the result of a long faradization. There was an immediate and profound diminution of intracranial volume accompanied by a considerable reduction of intrapleural pressure. As it was impossible to isolate and stimulate the whole nerve, it was impossible to obtain a tetanic contraction of the whole diaphragm. In consequence, small movements of both levers follow the first big movement. It will be seen in the tracing that the intracranial volume, although not remaining at its minimum point, does not return to its original size until the stimulation has ceased. This is probably to be attributed to the stimulation causing the diaphragm to remain partially contracted. During the period of stimulation, the arterial blood pressure rose, but there were no respiratory waves superposed on the tracing. No results were obtained from stimulation of the nerves with single shocks, but short periods of faradization yielded tracings similar to the one already described.

Time relations. Figure 7 is taken from a tracing recorded on a rapidly moving drum and permits a closer analysis of the time relations between the movements of the levers recording changes in the intracranial volume and in intrathoracic pressure, respectively. Careful measurement showed that the two levers reached their lowest points simultaneously. On the other hand, the commencements of these movements were not synchronous, the intrathoracic pressure being considerably lowered before any change in the intracranial volume could be detected. The expiratory phases, however, ran closely parallel to one another, with some tendency for the restoration to the normal of the intracranial volume to precede that of intrathoracic pressure. Other tracings have been similarly analyzed. The only appreciable difference between the findings in these and the findings already reported was that there may be a lag in the production of the maximum decrease of intracranial volume as compared with that of intrathoracic pressure. This was more noticeable in those cases in which the movements obtained were very large and may be, and very probably is, instrumental in origin, due to the inertia of the moving lever.

Influence of Arterial Pressure

At first it was generally found that, at the beginning of an experiment, even the most vigorous inspiratory movements coupled with considerable resistance to air entry produced only very small movements of the lever recording changes in intracranial volume or even none at all. Occasionally there might be a few movements of the lever, each smaller than its predecessor, and the whole dying away rapidly, although the resistance to breathing was still maintained (Fig. 8).

On the other hand, after the experiment had been in progress for some time, it was usually possible to induce quite large movements of the intracranial lever, using methods apparently exactly the same as those which had previously failed. In the first case, the animal was in good condition with normal blood pressure. In the second, the results of the operative interference had impaired its condition with, usually but by no means always, a lessened blood pressure. It was thought therefore that one of the essential factors was a lowering of the systemic blood pressure.

Vagal stimulation. The most obvious way of testing this hypothesis was to stimulate the peripheral end of a cut vagus nerve during a period of vigorous respiration with the tracheal cannula blocked. Experiments were performed on these lines and a tracing from one is reproduced as Figure 9. From this it will be seen that closure of the tracheal cannula produced small movements of brain-flap, exactly similar to those shown in Figure 8. When these had ceased, the peripheral end of the cut vagus nerve was stimulated with the faradic current. The blood pressure fell at once to about half its previous height

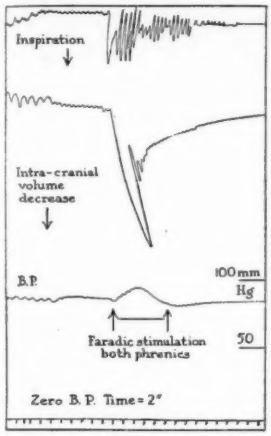

Fig. 6.

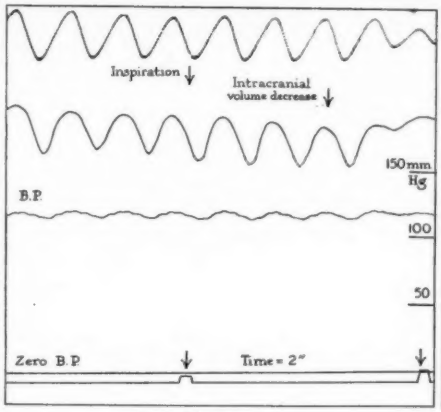

Fig. 7.

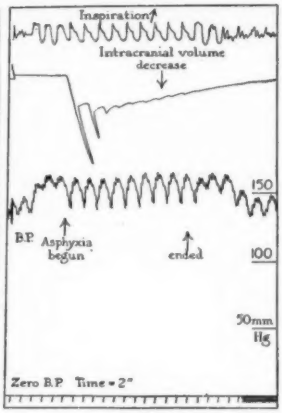

Fig. 8.

Fig. 6. To show the result of simultaneous faradization of both phrenic nerves. A rapid diminution of intracranial volume occurs simultaneously with a similar diminution of intrathoracic pressure, caused by the sharp descent of the diaphragm.

Fig. 7. This tracing was taken on a rapidly moving drum to permit analysis of the time relationships between the movements of the respiratory and volume levers.

Fig. 8. This shows the type of brain-flap which occurs occasionally at the commencement of an experiment. Coincident with the commencement of asphyxia, there is a sharp fall of the volume lever but, with each successive respiratory movement, the extent of the excursion of the volume lever is less, the whole dying away rapidly. This occurs in spite of the continuance of the resistance to breathing.

and as promptly recovered on the cessation of the stimulation. No sign of brain-flap was observed either during or after the period of stimulation. The whole experiment was then repeated with similar results.

The effect of histamine. Another possibility was that brain-flap does not occur without the coexistence of some degree of shock. Undoubtedly, the combination of anæsthetic, operative interference, exposure, etc., would be likely to cause this condition, which was probably always present to a greater or less extent after an hour had elapsed from the commencement of the experiment, at about which time brain-flap commonly made its appearance.

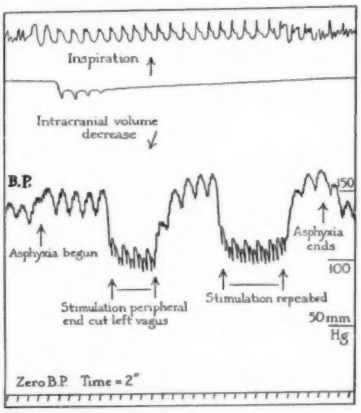

Fig. 9. This tracing shows, to commence with, a few movements of the volume lever, exactly similar to, but smaller, than, those in the preceding figure. When these had ceased, the peripheral end of the cut left vagus was faradized. The blood pressure fell considerably, but, although the tracheal cannula was blocked, there were no signs of brain-flap. A repetition of the experiment was also without result. In this tracing the respiratory lever moves upward in inspiration.

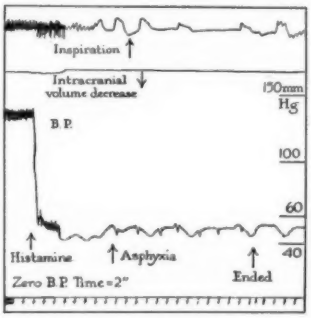

Fig. 10. To show the result of the injection of histamine. The usual profound fall of blood pressure occurred but, in spite of vigorous inspiratory movements, no trace of brain-flap could be seen. The respiratory tracing was taken with a stethograph, the lever moving upward in inspiration.

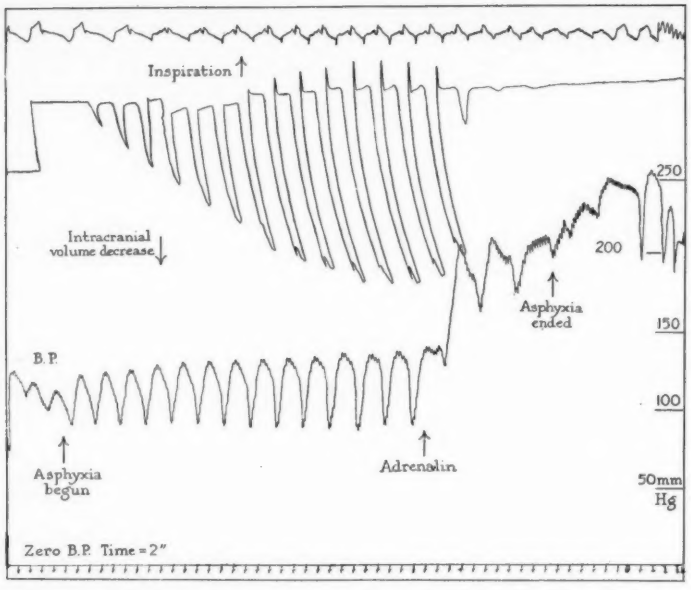

Fig. 11. Brain-flap having been induced by asphyxia, about 1 milligram of adrenalin was injected intravenously. The expected rise of blood pressure occurred and, with it, there was a complete cessation of brain-flap. Respiration was recorded by the stethograph.

Again, in the cases seen clinically and described earlier, shock must have been present, often in a very marked degree.

To imitate surgical shock, small doses of histamine phosphate (about 2 milligrams) were injected intravenously early in an experiment, when attempts to produce brain-flap by occlusion of the tracheal cannula had already been found to fail. A tracing, reproduced in Figure 10, displays the characteristic prompt and severe fall of systemic blood pressure which follows the intravenous administration of this drug. At the same time, occlusion of the tracheal cannula produced not the faintest sign of brain-flap.

Adrenalin. The most certain method of inducing this condition was found to be the administration, intravenously, of small doses of adrenalin (about 1 milligram). The usual immediate and pronounced effect on the systemic blood pressure followed, which might rise to 250 or 300 milligrams of mercury. When the immediate effects of the drug had passed off, the blood pressure fell to a level below that at which it had been before the injection. It was then found that, in an animal in which there had previously been no movements of the intracranial volume lever following occlusion of the tracheal cannula, brain-flap was readily produced by this method and that the excursions of the lever might be very large. Figure 11 is a tracing reproduced to display these points. Afterward the animal remained sensitive to this treatment for some time, usually until the end of the experiment. Occasionally, two injections of adrenalin were found necessary.

Adrenalin in sensitive animals. Further observations were then made on animals thus rendered sensitive to occlusion of the tracheal cannula. Brain-flap was induced in the usual manner and a further injection of adrenalin given. It was then found that, *pari passu* with the rise of the systemic arterial pressure, the variations in intracranial volume ceased, though there might be, and usually was, an increase in the total volume. While the blood pressure was rising or during the period in which it remained practically constant near the maximum, it was difficult or even impossible to induce brain-flap by occlusion of the tracheal cannula, however vigorous the inspiratory movements might be. When, however, the blood pressure had commenced to fall, because of the fading of the adrenalin action, the movements of brain-flap could be induced easily, but at a much higher level of blood pressure than that at which the movement ceased while the pressure was rising (Fig. 12). It was also observed that, if the rise of blood pressure was slow, as a result of the very gradual injection of adrenalin, the diminution in size of the movements of the intracranial volume lever was also gradual, varying more or less inversely as the rise of the blood pressure, as shown by the tracings in Figure 13.

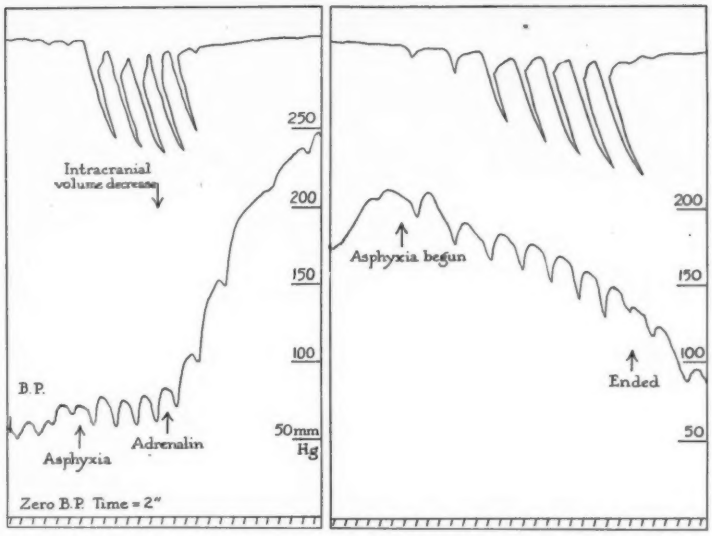

Fig. 12. The first part of this tracing shows the abolition of brain-flap by the intravenous injection of adrenalin, just as in the last figure. In the second part, however, it will be evident that, at a time when the systemic blood pressure was still at a height above that at which brain-flap had previously ceased though below its maximum, asphyxia was able to re-induce the condition. For exact synchronicity between the volume and blood pressure tracings, the latter should be transferred one-sixteenth of an inch to the left.

Influence of Interference with the Cerebral Circulation

At first, attempts were made to examine the results of occlusion of, on the one hand, the carotid arteries and, on the other, the external jugular veins, the internal in the dog being remarkably small or even absent. No definite results were obtained from this procedure alone. It was thought that this was probably due to the very efficient blood supply afforded by the vertebral vessels. In consequence, the method of ligating these vessels was employed, which was introduced by Sherrington (9) for the decapitation operation in cats. In this a piece of stout string is passed, by means of a packing needle, in front of the vertebral column and the ends brought out on the dorsal surface of the neck. The muscles of the back and sides of the neck are then divided and the string tightly knotted at the back, so that the vertebral vessels are compressed against the spinal column.

Occlusion of the carotid arteries. After both arteries had been freed from the other structures contained in the sheath, a ligature was passed loosely round each in such a manner that, when the ends were pulled the lumen of the vessel would be obliterated, without at the same time any risk of damaging its coats. On release of the ligature the blood supply to the brain was instantly restored. The natural result of occluding these arteries, after the vertebral vessels have been tied, is to stimulate the vasomotor center by the resulting anæmia of the medulla and so to cause a rise of blood pressure. This, in fact, did not always occur, probably because of the extensive anastomoses, described by Hill (3), between the intercostal and spinal arteries.

After the experimental animal had been rendered sensitive to occlusion of the tracheal cannula, the air entry was checked and, as soon as brain-flap was established, the carotid arteries were occluded, usually simultaneously. The result, in most cases, was a fall in intracranial volume, coupled sometimes with an increase in the size of the excursions of the lever. On releasing the ligatures, there was a rapid increase in the intracranial volume, together with a reduction or abolition of brain-flap (Fig. 14).

Sometimes, but not in this case, the volume lever ultimately reached a level higher than that at the beginning of the experiment, showing that the intracranial volume had undergone some more or less permanent increase.

It will be noticed that the arterial blood pressure rose to nearly 200 milligrams of mercury while the movements of brain-flap were still very large. This does not, in any way, antagonize the statements made that the action of adrenalin in causing a rise of blood pressure prevents or stops the development

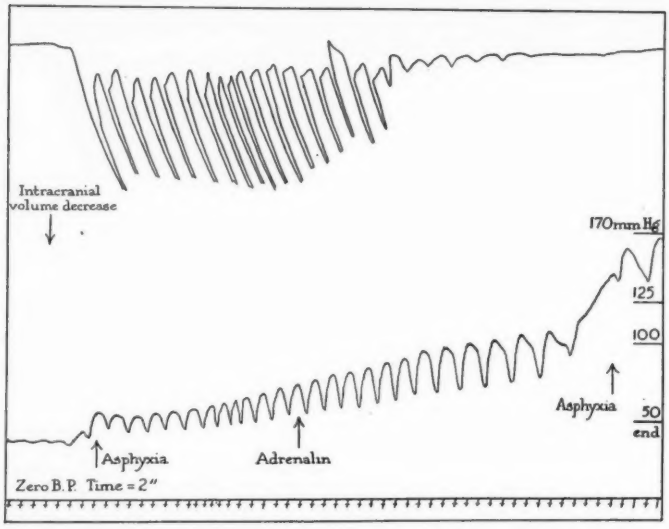

Fig. 13. To show the effects of a slow injection of adrenalin. The blood pressure rose slowly at first and with the gradual rise there was an equally gradual diminution of the size of the movements of the volume lever.

of brain-flap. Here the high blood pressure was confined to the extracranial part of the body and could not reach the cranial cavity except through the spinal arteries and any others which might anastomose with the carotid or vertebral arteries. The great rise of blood pressure alone suggests that there can have been only an inefficient anastomosis; for, had the blood supply to the medulla been more extensive, there would have been less stimulation of the vasomotor center.

Occlusion of the jugular veins. The jugular veins were prepared in a manner similar to that described for the carotid arteries. The result of their occlusion in an animal, in which the vertebral vessels had been tied and brain-flap induced by occlusion of the tracheal cannula, was to cause an increase in the intracranial volume and, generally, a reduction of the amplitude of the excursions of the lever. On release of the ligatures, the latter increased in size and there might be a reduction of intracranial volume, though this was not invariable (Fig. 15). In some cases, as in this, a large rise of systemic blood pressure occurred during the occlusion of the vessels and under such conditions the fall of intracranial volume on release of the ligatures was usually absent.

Influence of Variations in the Intracranial Pressure

Weed and McKibben (11) were the first to observe that intravenous injection of strongly hypertonic solutions such as 30 per cent sodium chloride and of hypotonic fluids such as distilled water were ollowed by a great decrease and increase respectively in the pressure of cerebrospinal fluid in the cisterna magna. These investigations have been repeated and confirmed by many other workers.

Experiments were performed with this method in an attempt to discover what relation if any might exist between the intracranial pressure and brain-flap. It was found, however, that so many other complications were introduced that it was thought that this part of the work should form the subject of a second paper. It is sufficient, at present, merely to state that the largest movements of brain-flap seen in the whole course of this work, were developed after the injection of hypertonic saline.

Other Experimental Observations

So far the description of the experimental work has been practically confined to the rapidly occurring decreases and increases in the intracranial volume, called here for the lack of a better name brain-flap, which is induced by, and follows closely on, the respiratory movements of a trephined animal, in which air entry to the lungs is prevented.

Besides these, other rather grosser changes in the intracranial volume have been observed to occur. Reference to Figures 1 and 15 will show that, in addition to the condition of brain-flap, there is a second variation. At first there was a progressive lowering of the point to which the lever returned on each expiration until a maximum was reached. This shows that the lowering on inspiration of the intracranial volume was not entirely made up with each expiration and that this persisted until the obstruction to breathing was removed.

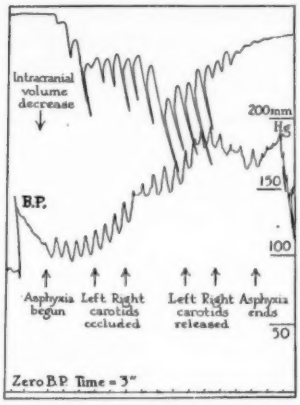

Fig. 14. To show the effects of occluding the carotid arteries, the vertebrals being already ligated. When asphyxia was started, there was a small reduction in intracranial volume. When this had reached a steady level, the two carotids were occluded. No change followed the blocking of the first but, on closure of the second, there was a further diminution of intracranial volume, with an increase in size of the brain-flap excursions. On release of one artery, the intracranial volume began to increase and, on release of the second, this became more rapid and was associated with an almost complete cessation of brain-flap. The rise of systemic pressure is due to anæmia of the vasomotor center in the medulla. The blood pressure tracing should be transferred one-eighth of an inch to the left to obtain synchronicity with the volume record.

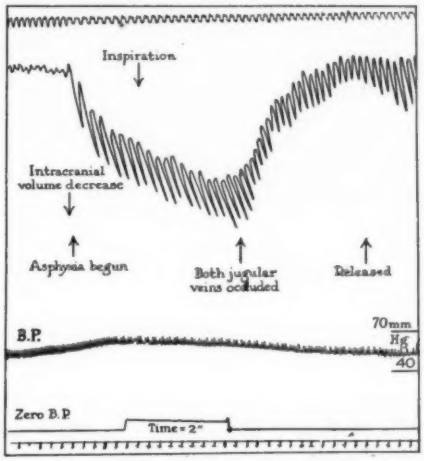

Fig. 15. To show the effects of occlusion of the jugular veins, the vertebral vessels having been previously tied. Brain-flap having been established, there was a very large reduction of intracranial volume. Occlusion of the jugulars was followed by a prompt return of the intracranial volume to its original level, with a reduction in size of the excursions of brain-flap. On release of the vessels, the volume movements became larger, but there was no repetition of the general fall of level.

Adrenalin, as has already been stated, causes the movements of brain-flap to cease. Not only does it do this but usually causes, in addition, the intracranial volume to increase.

Although no change of intracranial volume is shown in Figure 10 following the injection of histamine, a diminution usually occurred and might be large. On one occasion it was very well marked, the volume and blood pressure recorders falling almost parallel to one another.

On a few occasions, over-vigorous artificial respiration by the pump has caused, in a sensitive animal, the inverse of brain-flap. In this there has been an increase in the intracranial volume with each positive (inspiratory) stroke of the pump. When the pump was disconnected from the tracheal cannula, there was a prompt cessation of these movements and a reduction in intracranial volume. Further, when the tracheal cannula was blocked, true brain-flap was produced, the intracranial volume falling with each inspiratory movement.

Figure 16 has been prepared to show the results of forcible compression of the heart in the dead animal. Immediately after death the chest was opened and the heart squeezed vigorously with the hand. Each compression was followed at once by a rise in the intracranial volume, succeeded by a return of the lever to the base line on relaxing the grip.

A somewhat similar result was given by blowing strongly into the pleural cavity of an animal, immediately after death, through the tube inserted for the purpose of registering the changes of pressure within that cavity.

DISCUSSION

At the outset, it must be emphasized that the clinical observations and experimental results here reported do not, and are not intended to, represent a state of affairs occurring in the normal condition. The mere fact that the skull has been opened and the dura exposed to atmospheric pressure during the whole course of the observations is at once fatal to any such suggestion. It is, in our opinion, in the highest degree improbable that any such variations in the cerebral volume as are here described can occur in the animal with an intact skull.

It was suggested by Monro (6) about 150 years ago that the bony skull could be regarded physiologically as a rigid box, and this view has met with general approval. Except in so far as there may be a possibility of forcing cerebrospinal fluid for a very short

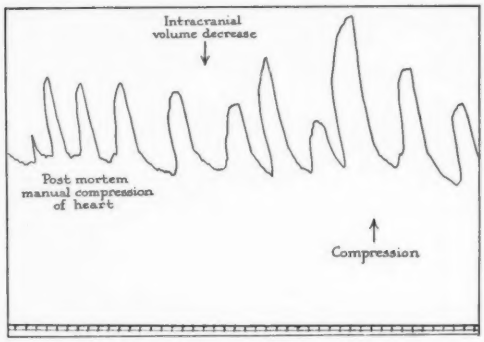

Fig. 16. This tracing shows the changes of intracranial volume which follow on manual compression of the heart in a recently dead animal. Each act of compression is followed by an increase in the volume, to be succeeded by a reduction on the release of the compression. This is the inverse movement to that of brain-flap.

distance along the nerve sheaths as they issue from the skull, it would seem that there can be no change in volume of the total intracranial contents and that such changes as are here described could never occur so long as the bony cranium remains intact.

Under the conditions of experiment as here described, it is clear that the dura mater may, on the one hand, fall into a considerable extent and, on the other, bulge outward slightly until the limit of elasticity of the membrane is reached. It is with such changes in volume occurring very rapidly that we are concerned.

In the first place we have to consider which substance of those present within the skull can undergo such rapid changes. It is inconceivable that the solid matter, the gray and white matter of the brain, can change in volume with such rapidity. There is little room for doubt that the cerebrospinal fluid, secreted by the choroid plexuses and absorbed into the veins through the pacchionian bodies, may vary in volume from time to time. It is, however, in the highest degree improbable that these processes of absorption and secretion should take place with the rapidity of the movements of brain-flap and that they should have, when present, that time relationship with the respiration which is also characteristic of brain-flap.

There remains the content of blood within the skull. This quantum, whatever it may actually be from its nature as part of the blood in general circulation, is eminently variable. The tracing shown as Figure 16 shows clearly that changes of intracranial volume may be due to variations in the blood content. On compression of the heart immediately after death, the volume lever attached to the skull rose and, on relaxation of the compression, it fell to its original level. Here there can be little doubt that compression of the heart had forced blood back through the practically valveless great veins (3) of the neck into the cranial cavity. There was no complication due to respiration, artificial or natural, to confuse the issue. Although it is true that the movement shown in the tracing is the exact inverse of that seen in brain-flap, this is of no importance. The tracing shows that variations of blood volume in the cerebral circulation can produce marked changes in intracranial volume.

We may conclude then that brain-flap is dependent on variations of the content of blood within the skull. We have now to consider what factors are, or may be, concerned in the production of these variations. The volume of blood within the skull from moment to moment must depend on the relative rates of inflow and outflow. There are three possible conditions: that in which the inflow equals, is greater than, or is less than the outflow.

In the first case the quantity of blood in the skull will remain constant and there will be no brain-flap. This is the usual condition, unless special measures are taken to upset the balance between inflow and outflow.

In the second condition, with inflow greater than outflow, there will be a tendency for the intracranial tension to rise, and this will be displayed under these experimental conditions by a bulging of the dura mater through the trephine opening with a rise in the volume lever. This was found to take place in an experiment in which all exit of flow from the skull was prevented, while the carotid arteries remained open. This was done by temporary occlusion of the jugular veins, the vertebral vessels having been previously tied (Fig. 15). No brain-flap will arise from this procedure alone.

In the third case, with inflow less than outflow, there will be a tendency for the intracranial pressure to fall and, under our experimental conditions, this will appear as a sinking in of the dura mater and a fall in the volume lever. This was found to be the case when the carotid arteries were temporarily blocked after the vertebral vessels had been tied (Fig. 14). This *by itself* will not give rise to brain-flap.

The condition of brain-flap is characterized by a rapid alternation of the conditions present in the second and third cases, a rapid fall and rise of the volume lever, the excursions of the lever in each direction being equal or nearly so, with a tendency for the upward movement to be less than the downward. This may be stated as a rapid alternation of short periods of time in which inflow is less and greater than outflow.

We will next consider the conditions governing the flow of blood into and out of the cranial cavity. On the side of inflow, there is the systemic blood pressure opposed by the resistance of the arterioles and capillary bed. To cause outflow there is the venous pressure, practically the resultant of the two former forces, aided or opposed by the changes in intrathoracic pressure during inspiration and expiration. At the same time, it should be remembered that the whole intracranial circulation is subject to the almost constant pressure of the cerebrospinal fluid, a pressure of the same order as, and varying slightly in either direction from, that in the veins (1).

If the experimental observations are considered, it will be clear that they may be divided into two groups, those in which brain-flap did and did not appear, respectively. It may be observed here that, in most respects, there was no difference in the experimental procedure employed in both cases. The former group will be considered first.

In all the experiments in which brain-flap was produced, there was only one constant factor, excessive aspiration by the thorax. Hyperpnœa alone, even when very vigorous, was not sufficient to induce the condition (Fig. 1). Hyperpnœa, if the air entry to the lungs be perfectly free, as it is with a large-bored tracheal cannula, will produce but little change in the intrathoracic pressure and, so, little change in the aspirating action of the chest in inspiration on the blood contained in the thin-walled great veins. On the other hand, if the lungs cannot follow freely the movement of the chest wall in inspiration, the pressure in the pleural cavity will be greatly reduced and the aspirating effect correspondingly increased. If then the tracheal cannula be blocked at the end of expiration, the result of even normal breathing will be to cause considerable diminution of the intrapleural pressure on inspiration with a return to the previous pressure on expiration. This will produce a great increase in the aspirating effect during inspiration and a rapid decrease during expiration. This should, and under the right conditions does, induce that type of brain-flap, in which the movements are large from the beginning (Fig. 5, the first part). Again, it would be expected that if the tracheal cannula be blocked at the end of inspiration there would be less tendency for the intrapleural pressure to be further reduced with succeeding inspirations, until, on the one hand, accumulation of carbon dioxide in the blood caused the respirations to become deeper and, on the other, the continued abstraction of oxygen by the blood from the lungs had reduced their volume and the intrapleural pressure. Under the right conditions brain-flap in which the movements should be small at first and gradually increase in size would thus be produced (see Fig. 5).

Stimulation of the phrenic nerves on both sides simultaneously leads to a sharp descent of the diaphragm and, with the tracheal cannula blocked, to a sudden diminution of intrathoracic pressure. Experiment showed, again, that this diminution with its aspiratory effect produced an equally rapid reduction of brain volume (Fig. 6).

Even more conclusive proof of the dependence of brain-flap on respiration was obtained by those experiments in which the central end of one vagus was stimulated, the other nerve being left intact (Fig. 2). While brain-flap was being maintained as a result of occluding the tracheal cannula, stimulation of the nerve was commenced. This caused respiration to cease during the period of stimulation. Brain-

flap ceased with the cessation of respiration and recommenced with the respiration on the termination of the period of nerve stimulation. Here there was only one difference between the periods with and without brain-flap, the fact that the respiration, and with it the aspirating effect of the thoracic movements, had ceased. The blood pressure remained practically constant throughout.

Consideration of the time relations between the movements of the intracranial volume and of the intrathoracic pressure levers (Figs. 4 and 7) shows that both reach their lowest points simultaneously. This means that the maximum diminution of intracranial volume occurs at the same moment as the maximum reduction of intrapleural pressure or the maximum aspirating effect. This confirms the dependence of brain-flap on this action of the thorax. At the same time, it disproves the suggestion which might be made, that these changes in intracranial volume are the result of exaggerations of the normal respiratory waves found in the arterial blood pressure tracing. It is true that these waves are exaggerated by the occlusion of the tracheal cannula, as would indeed be expected, and this may be seen to be the case in Figures 9 and 11. It is, however, well known that the arterial pressure rises during the last two-thirds of the inspiratory phase to reach its maximum at the end of the first third of expiration. Now, with a rise in arterial pressure the intracranial volume rises and, were brain-flap due to an exaggeration of the respiratory waves on the blood pressure, there should be an increase in intracranial volume reaching its maximum at a point in close proximity to that at which, in fact, it is at a minimum.

We have now considered fully the relationship between brain-flap and one of the factors concerned in the regulation of the outflow from the cerebral circulation, the aspirating action of the thorax. Little has been said with regard to the damming back of blood which occurs with expiration except in so far as it is responsible for the return of the volume of the cranial content to its previous position. It must be remembered that the dura mater is a very tough, inelastic membrane which will offer considerably higher resistance to a force tending to cause it to expand than will be the case with the thin walled veins. The other factor in connection with the outflow from the brain is the venous pressure. This is so dependent on the arterial blood pressure that it needs no special treatment.

The effects of variation in the resistance to the flow of blood offered by the cerebral arterioles and capillary bed need hardly be considered. It is well known that the arterioles are very badly supplied with smooth muscle fibers. Again, Roy and Sherrington (8) have shown that these vessels in experimental animals are devoid of a vasomotor nerve supply. In consequence, they will respond only very slightly, if at all, to the usual agents causing constriction or relaxation of these elements. Roy and Sherrington found that the circulation in the brain was directly determined by the systemic blood pressure.

The relationship between the systemic blood pressure, the more important factor concerned with inflow of blood to the cerebral circulation, and brain-flap must now be considered. This condition has been induced with arterial pressures, as measured in the femoral artery, ranging from 140 to 40 milligrams of mercury, indicating that there is little connection between the actual height of arterial pressure and occurrence of brain-flap.

On the other hand, it was found that the great rise of blood pressure caused by the intravenous administration of adrenalin would put a stop to brain-flap induced by occlusion of the tracheal cannula, although the interference with respiration was still maintained. This suggests that a very high blood pressure is incompatible with brain-flap.

Further experiments, however, showed that, when the blood pressure had fallen somewhat from its highest point but was still above that at which brain-flap had previously ceased, the condition could again be induced by occlusion of the tracheal cannula. From this, it would seem that the mere height of blood pressure is not alone the cause of the cessation of the movement.

In order to arrive at an explanation of these apparently contradictory findings, it will be necessary to consider what changes will occur in the cerebral circulation as a result of the

administration of adrenalin. It has been shown that in the animal with the skull intact, the effect of adrenalin is to raise the arterial, venous, and cerebrospinal fluid pressures (1). Our own experiments show that, besides the cessation of brain-flap, there is an increase of the volume of the intracranial contents. This must mean that blood is being forced into the skull at a rate greater than that at which it can escape. If blood is already leaving the skull at the maximum possible rate, as must be the case under these circumstances, aspiration by the thorax cannot increase this rate and in consequence brain-flap should cease, as in fact it does.

It has been shown by Becht (1) that, after the administration of adrenalin, the venous pressure rises in the cerebral sinuses with the systemic arterial pressure and is maintained at a relatively high level for some time afterward, when the latter has fallen considerably. Although the venous pressure remains high, the fact that the arterial pressure has fallen, points to a diminished inflow of blood. If this is the case, it is evident that aspiration will be able to withdraw blood at a more rapid rate than that of the previous outflow, while expiration will check the process. As a result of these two factors, brain-flap will occur. Further, the extent of the movements should increase with a further fall of arterial pressure (see Fig. 12).

Additional support is lent to this argument by the facts recorded in Figure 13. Here adrenalin was injected, at first very slowly in dilute solution and, later, more rapidly. The systemic blood pressure at first rose very slowly and with it the extent of the excursions of brain-flap became progressively smaller. As the arterial pressure rose, the rate at which the blood entered the sinuses would be increased so that aspiration would have less and less effect, thus causing the reduction of brain-flap.

To sum up this section of the discussion, it may be said that, in those cases in which it was possible to induce brain-flap experimentally, there was one constant feature—excessive aspiration by the thorax, produced by blocking, partially or completely, the air entry to the lungs. It seems that, up to a point, the height of the arterial pressure has little effect; but that once this point is passed the rate of outflow from the skull reaches its maximum and can no longer be increased by aspiratory action by the thorax.

So far attention has been confined to those cases in which brain-flap was successfully produced by experiment. The very numerous cases in which the condition could not be induced have now to be considered.

In every experiment performed, there was at the beginning a period in which even the most vigorous inspiratory movements of the thorax, coupled with complete closure of the tracheal cannula, failed to produce brain-flap. At most, and then only rarely, a few small movements, rapidly becoming smaller and soon disappearing altogether, were obtained (Fig. 8). Later on in the experiment we had no such difficulty; in one experiment alone did we fail at all times to obtain brain-flap.

There can be no question as to the occlusion of the tracheal cannula. The orifices were closed with finger and thumb in the same manner exactly in each case.

Lowering blood pressure by vagal stimulation with complete closure of air entry was entirely without avail. Similarly, administration of histamine, in order to lower blood pressure and induce a condition closely akin to, if not identical with, surgical shock was equally useless. The sole result of the latter procedure, not invariably, was to cause a reduction of intracranial volume. Again, the raising of the blood pressure by the injection of adrenalin was without immediate effect.

In these experiments we attempted to vary the rates of inflow and outflow of the cerebral circulation in all possible ways. Stimulation of the peripheral end of the vagus causes a reduction of blood pressure and so a decrease in inflow, with possibly an increase in the venous pressure in the sinuses. Histamine produces a lowering of both arterial and venous pressures in the skull as has been shown by Lee (5). This should produce a reduction of both inflow and outflow. Finally, adrenalin, as already stated, causes an increase in both inflow and outflow.

As explained in the experimental section, the only reliable method of inducing the con-

dition was found to be the injection of adrenalin. Sooner or later after this had been done, it would be found that brain-flap could be induced. A point of some slight interest as showing the small importance of the arterial pressure in connection with the phenomenon is the fact that in several experiments brain-flap was induced some time after the administration of adrenalin with the blood pressure standing at exactly the same figure as that at which it had stood earlier when all attempts to induce brain-flap had failed.

It is clear from a comparison of the experiments that failed at first and later were successful, that some change must take place in the conditions of the cerebral circulation. At first there is no brain-flap, or only a very slight manifestation of it which soon disappears. This means that, in the normal animal, inflow and outflow of blood to and from the cerebral circulation remain exactly balanced, however great may be the variations in the aspirating action of the thorax. Later on, this balance becomes upset and brain-flap develops. The mechanism and nature of this change is at present obscure. There seem however to be three possibilities in this connection. The first is that outflow should be at its maximum to commence with so that aspiration by the thorax cannot further increase the rate of discharge. This seems improbable in view of the facts that, first, aspiration apart, the principal, if not the only real, factor concerned with the pressure in the sinuses is the arterial blood pressure, and, second, at two different stages in the same experiment, the blood pressure may be exactly the same and yet at one stage it may be impossible to induce brain-flap and at the other the condition may appear readily. The second possibility is that there may be some, as yet unknown, mechanism which so controls the inflow as to make it exactly equivalent to the outflow at all times, however, much the latter may vary. Nothing is known of such a mechanism and, having regard to the circumstances of the cerebral circulation, it seems improbable that such should exist. The third possibility is based on the facts that (a) the only certain way of inducing the condition in animals was found to be by the administration of adrenalin, and (b) it is known that the venous pressure remains high for some considerable length of time after the return of the arterial pressure to the normal. It may be that the arterioles become, as it were, strained by the passive pressure exerted on them by the systemic blood pressure and remain dilated throughout the experiment, thus enabling the venous pressure to remain high with a relatively low arterial pressure.

Again, it may be that variations in the intracranial pressure may condition this change in the cerebral circulation. At present our experiments in this direction are not sufficiently complete to allow us to say anything on this point, and this part of the subject is reserved for a second publication. Hill (3) states that a high intracranial pressure would tend to damp any other variations in pressure or volume within the cranial cavity.[1]

Finally, it remains to consider what, if any, similarity can be traced between these experimental results and the clinical findings.

Our experiments may be divided into three classes: (1) those with no brain-flap, (2) those in which brain-flap was represented by a few movements of the intracranial volume lever, soon vanishing entirely (Figs. 8 and 9), and (3) those in which brain-flap was easily induced when desired.

The clinical observations may be divided into three exactly similar classes. The absence of references in surgical literature to phenomena of this kind can mean only one of two things—either brain-flap is so common as to be unworthy of remark or so rare that it has been observed only on a few occasions. Considering the frequency of trephining as an operation and the rarity with which this condition has occurred in our experience, it is clear that the second explanation is correct. Stertorous breathing is frequently associated with the conditions for which trephining is performed and yet brain-flap is practically unknown. Therefore, the ordinary trephine operation in a case in which there is some obstruction to breathing forms an exact parallel to our first class of experimental findings.

[1] In this connection, it may be observed that we have very suggestive evidence from the clinical side, which we hope to confirm by experiment. This clinical evidence was obtained only recently after the compilation of this paper.

Reference to the first section of this paper will show that in our fourth and fifth cases there was a transient appearance of brain-flap. The movements were associated with deep respirations but soon disappeared. These two cases may be considered as similar to, or even identical with, our second class of experimental findings.

Our first and third clinical cases, and probably Wheeler's case should be joined to them —displayed movements of the brain associated with difficulty of breathing, which continued for some time. They are sufficiently parallel to our third class of experimental findings to permit us to suggest that there must have been similar causes at work in the two conditions. Only one important objection seems possible, that, in the clinical cases, the condition arose apparently spontaneously, but, in the experimental work, only with active interference. This objection we think, is not well founded. It is true that, in our later experimental work, care was taken to block the tracheal cannula completely. This was done to give us a firm basis for our work. In the earlier experiments, it was observed that brain-flap could be produced by far less vigorous interference with the air entry, such as might occur with excessive mucus in the trachea, though the movements were then less pronounced. It is, of course, impossible to say how much or how little resistance there was to breathing in our clinical cases and consequently how much change in the intrathoracic pressure would occur on inspiration. We feel that, with the closely parallel results that have occurred in the operating theater and the experimental laboratory, we are justified in claiming that we have reproduced in dogs a condition exactly similar to the rare occurrences of brain-flap observed clinically.

SUMMARY

1. A phenomenon of extensive changes in intracranial volume, synchronous with respiration, in man, with the experimental production of a similar condition in dogs, is described under the term "brain-flap."

2. In an animal just after death, great changes in intracranial volume can be produced, after the chest is opened if the heart is alternately squeezed and relaxed. These charges from the mechanical conditions must be opposite in sign to those of brain-flap.

3. The condition is dependent on changes in the intracranial content of blood.

4. The volume of a brain which exhibits this condition diminishes with inspiration and increases with expiration.

5. Dyspnœa is a constant factor in its production, both clinically and experimentally.

6. In the presence of dyspnœa, the condition may occur with systemic blood pressures ranging from 140 to 40 millimeters of mercury.

7. On the other hand, dyspnœa may fail to produce it with systemic blood pressures as low as 40 millimeters of mercury.

8. Brain-flap is cut short by the marked rise of arterial blood pressure, caused by intravenous injection of adrenalin.

9. *Within fairly wide limits*, brain-flap seems not dependent on arterial pressure.

10. The immediate cause of brain-flap would appear to be an imbalance between the quantity of blood entering and leaving the cranial cavity from moment to moment.

11. It is certain that one, and probably the main, factor in this imbalance is the excessive aspiration of blood from the veins by the chest during dyspnœic respiration. It is probable: (a) that a relatively deficient inflow to the cranial cavity from the arterial side may be an essential factor; and (b) that while a high pressure of the cerebrospinal fluid will damp the movements of the brain, a fall in pressure may permit them to appear.

Much valuable assistance in these experiments was given by Dr. K. Samaan, M.Sc., Ph.D., and the authors wish to acknowledge their debt in a spirit as wholehearted as that which prompted the aid.

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CLINICAL SURGERY

FROM THE ORTHOPEDIC SERVICE OF THE MASSACHUSETTS GENERAL HOSPITAL

FUSION OF THE SPINAL COLUMN

By NATHANIEL ALLISON, M.D., F.A.C.S., Boston

An operation, the purpose of which is to create bony union between several vertebræ is indicated when either injury or disease has caused or is causing a destruction of one or more vertebral bodies. It is also indicated when the spinal column is curving laterally as a result of paralysis of the muscles or of congenital defects in the bones.

The lesions which present the chief indication for spinal fusion are: tuberculosis of the adult spine, compression fracture of the spine, scoliosis which results from poliomyelitis, and scoliosis due to congenital bone defects.

DANGERS AND POSSIBLE COMPLICATIONS

The operation[1] demands a thorough exposure of the posterior spinal arch throughout an extensive area.

The chief danger is that of surgical shock or operative infection. Infection is a most serious and menacing danger, against which every precaution in the way of careful operative technique and complete asepsis of technique is to be taken by the surgeon. There is little danger of serious hæmorrhage and little danger of damage to vital structures, provided the operation is carefully done.

PREPARATION OF THE PATIENT

The patient should be given rest in recumbency for several days before operation is undertaken. During this period he should receive the maximum of quiet and restful sleep, with light diet and free catharsis. The bed should be firm and unyielding, and only one pillow should be used.

The operative fields are the entire back and the tibial surface of one of the legs. The back should be shaved, scrubbed with soap and water, and cleansed with ether and alcohol the day before operation. The leg should be prepared in the same way.

The choice of anæsthesia rests with the surgeon. Ethylene or ether seem to us to be best suited for this operation.

The patient is placed face down on the operating table. To secure comfortable breathing in this position and to lessen surgical shock, the use of the cerebellar table is recommended (Fig. 1).

The back and one leg are exposed and painted with iodine. The leg is flexed at the knee, so that the heel rests near the buttock of the same side (Fig. 2). This exposes the anterior tibial surface. The table and leg are draped with sterile sheets. It is quicker and there is less consequent shock, if the two operative procedures are carried on simultaneously (Fig. 3). The operator and his assistant prepare the spine for fusion while the second operator and his assistant remove the graft from the tibia.

STEPS OF THE OPERATION

1. An incision is made in the midline over the tip of the spinous processes. The area of the spine to be fused has been determined by previous study of roentgenograms. Two vertebræ on each side of the vertebræ principally involved should be included. This means, that provided the tenth and eleventh dorsal are chiefly involved, then the fusion should include the eighth and ninth above and twelfth dorsal and first lumbar below. The incision therefore should expose the spinous processes of the eighth dorsal to second lumbar vertebra, inclusive.

2. After the division of the skin by vertical incision directly over the tips of the spinous processes, the posterior spinal ligament is exposed. The tips of the spinous processes are clearly seen and felt. The ligament should be divided accurately in true vertical line in the middle of each spinous process. This is best done by securing each spinous process in the grasp of a curved clamp, and making a vertical knife-cut in the

[1] This description briefly covers the method of spinal fusion in use at the Massachusetts General Hospital. No claim to originality is made. The method represents in part the technique first described by Hibbs and known as the Hibbs operation. The added step of osteoperiosteal graft implantation is done with the purpose of making fusion more certain between the vertebræ. In the hands of the several operators on the Orthopedic Service, this technique has yielded satisfactory results.

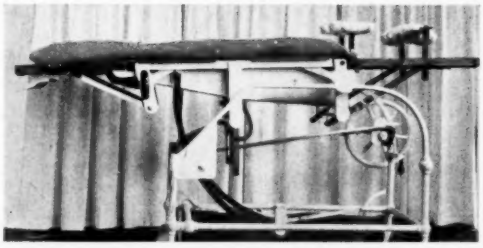

Fig. 1. The cerebellar table, which consists of a head rest and two shoulder supports. If the patient is placed face downward on the table, as is necessary in the spinal fusion operation, surgical shock is increased by the fact that each forcible movement on the spine itself is transmitted to the patient's abdomen. By the use of the shoulder supports and the head rest, the weight of the operation is transferred to the shoulders, thus protecting the abdomen and chest. Another valuable feature of this table is that it holds the spine in a straight position.

Fig. 2. The patient is placed on the cerebellar table with the head supported. The anæsthesia is started and the back and the leg are prepared for the operation.

mid-saggital plane of the spinous process (Fig. 4). From this entrance point the muscles, fascia, and periosteum may be stripped from the side of the process and along the laminæ below. The splits in the processes are united by a longitudinal incision throughout the region to be exposed. (Fig. 4, A).

By blunt dissection with a periosteal elevator (Fig. 5, A) and by gauze packing (Fig. 5, B), the entire posterior arch of the spine is then exposed. Retraction is accomplished by deep retractors, and later when the posterior arch of the spine is exposed by self-retaining retractors (Fig. 5, B).

3. *Preparation of bony bed.* From the surface of each lamina down as far as the lateral articulation, a bone shaving is turned up with a curved chisel, the lateral articulation is entered with the chisel and small curette, and the articular cartilage of the articulation removed. Each spinous process is split longitudinally into 3 fragments with a chisel, and the spinous process is cut across at its base, so that these fragments may be broken down to lie in position overlapping the space between the spinous process above and the spinous process below, thus bridging the entire area of the posterior spinal arch with bone fragments, each of which is still loosely attached to a base. All of these fragments are flattened out so that the surface is a rough surface for fractured bone fragments, continuous from the upper limit to the lower limit of the area to be fused. On each side of the midline, a osteoperiosteal graft from the tibia is now placed, bone side down: two strips of osteoperiosteal material ½ inch wide and long enough to reach the limits of the denuded area.

Bleeding is checked and controlled by tight packing, narrow gauze strips being used.

4. *Suture.* The edges of the supraspinous ligament are sutured together over the entire area with interrupted sutures of 30-day chromic catgut No. 2, or with silk sutures. The skin wound is closed with interrupted sutures of silk.

REMOVAL OF THE OSTEOPERIOSTEAL GRAFT

While one operator is preparing the bony bed on the posterior side of the spine, the second operator removes the osteoperiosteal graft. He has as a rule time to do this in a leisurely manner, as it takes longer to prepare the fusion bed. He should not anticipate the preparation of the spine because it is a valuable piece of technique to transfer the graft immediately to its future bed without delay and unnecessary handling. The graft should be cut free at the moment the spinal bed is ready and at that time transferred.

1. Incision is made from the tibial tubercle to a point 2 inches above the internal malleolus: the

Fig. 3. One operator has the spine as his field of operation. A second operator is preparing to remove the tibial graft. The leg is held flexed by the use of a sterile bandage, which is passed around the end of the table.

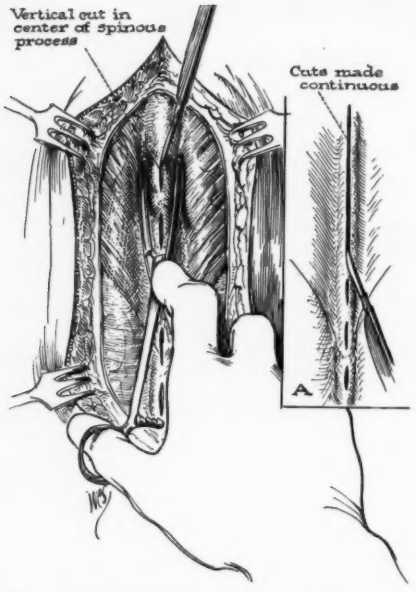

Fig. 4. After the exposure of the spinous processes, each spinous process must be divided directly in the midline. This is accomplished by holding the spinous process in the jaws of a curved clamp so that its outlines are well made out.

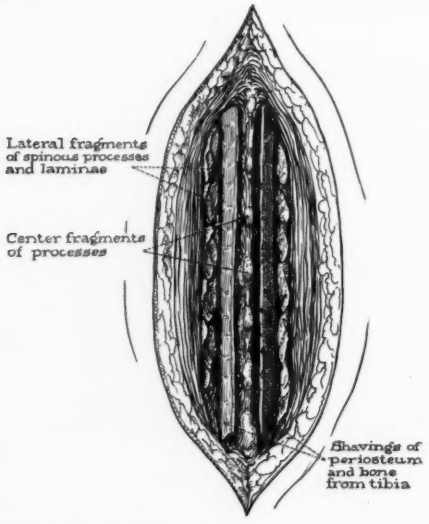
Lateral fragments of spinous processes and laminae
Center fragments of processes
Shavings of periosteum and bone from tibia

Fig. 6. After the spinous processes have been split and displaced and the posterior arch transformed into a denuded raw surface of the freshly fractured bones, two strips of osteoperiosteal tissue removed from the tibia are placed in this bony bed as transplants.

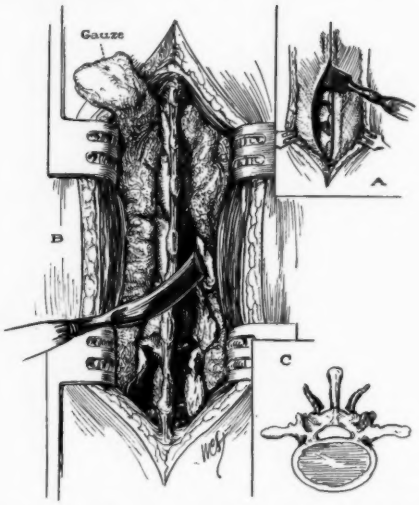

Fig. 5. A, Periosteum and muscles pushed from lateral surfaces of spinous processes and laminæ. B and C, Spinous processes split into three fragments; lateral fragments displaced outward and downward, middle fragments fractured at base and displaced directly downward.

entire subcutaneous surface of the tibia is exposed. It is well to mark out the graft with 2 longitudinal incisions made with a knife through the periosteum well into the cortical bone. The graft should be 1 inch wide and should be long enough to reach over the entire spinal area, i.e. it should be, as a rule, about 6 to 8 inches in length—this may be definitely decided by the use of a measuring probe supplied by the operator at work on the spine.

A sharp chisel the width of which is exactly that of the marked out graft is started at the upper end; with a mallet this chisel is engaged in the bony surface of the tibia, and by repeated light taps with the mallet, a shaving is rolled up (Figs. 7 and 8). It is important not to cut out with the chisel, but to keep the edges of this instrument engaged in the subperiosteal bone. As a result, the shaving removed should have periosteum on its upper surface and its under surface should be made up of a layer of subperiosteal bone $1/16$ to $1/32$ inch in thickness. This strip, 1 inch wide, is then cut longitudinally, making 2 strips $1/2$ inch wide, and these should be transferred to the first operator for implantation in the bony bed.

2. The wound over the tibia is sutured with interrupted silk stitches, and a tight compression bandage applied.

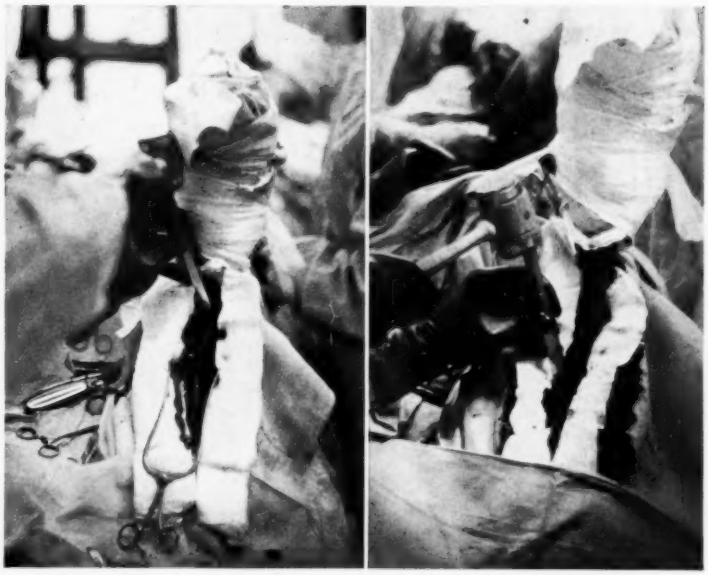

Fig. 7 (left). The removal of the osteoperiosteal graft. The subcutaneous surface of the tibia is exposed. The chisel is started at the lower end through the periosteum into the subperiosteal bone.

Fig. 8. The removal of the osteoperiosteal graft. By gentle blows with the mallet, a shaving of osteoperiosteal material is turned up from the anterior surface of the tibia. This is divided into two pieces which are transferred to the spinal field for implantation.

POSTOPERATIVE CARE

As soon as the operation is over—it should be done in about 1 hour or less—an estimate of the patient's general condition should be made: pulse rate and blood pressure compared to pulse rate and blood pressure before. The anæsthetic chart is very important, because the operation has peculiar possibilities of creating surgical shock. In all instances whether or not shock is evidenced, the patient should have shock treatment. He should be returned to bed as soon as possible; heat, elevation of foot of bed, shock enema, forced fluids, subdermal fluid if necessary, and morphine in sufficient quantity should be employed. If at the end of several hours there is evidence of postoperative shock, blood transfusion is indicated and 500 cubic centimeters of suitable blood should be given. The blood donor should be available during the first 24 hours after spinal fusion is performed. We have not found it necessary to place these patients on frames or in apparatus such as braces or plaster-of-Paris shells. A firm mattress is all that is necessary for the first 10 days. The use of pillows should be avoided.

After the removal of the sutures, and when wound healing is established, the patient should be supplied with a suitable brace or a plaster-of-Paris shell.

Absolute recumbency is necessary for a period of from 8 to 12 weeks. The patient should not be allowed up until a suitable jacket of plaster, leather, or celluloid, or a proper metal brace has been adjusted. The period of being up and about is gradually increased from ½ hour to full activity. Support and protection of the spine as well as carefully planned supervision of activity is necessary for at least 1 year after the operation.

FROM THE GYNECOLOGICAL CLINIC OF THE UNIVERSITY OF GHENT

INTRA-ABDOMINAL INCISION OF THE VAGINAL VAULT FOR DRAINAGE

By Dr. G. BAETEN, Ghent, Belgium
Assistant to Dr. Frans Baels, Cancer Department of the Gynecological Clinic of the University of Ghent

OUR modern aseptic and antiseptic methods have made the use of extensive drainage less common than formerly. Even in a severely infected case, it is advantageous to close the abdomen completely, although if hæmorrhage persists or the wound surface is abraded and necrotic tissue remains, one will not hesitate to make use of extensive drainage.

Extensive drainage is to be recommended chiefly after ablation of the adnexa in the presence of many adhesions, after operations for carcinoma of the cervix, for hæmatocele, or for adherent necrotic tumors. When it is only a matter of arresting a hæmorrhage, pressure can be produced with a tampon and the drain can be brought outside along the lower part of the abdominal incision, so that it may be removed after 24 to 36 hours. In such cases we always inject paraffin oil mixed with 10 per cent oil of camphor.

However, when the wound surface is large or considerable necrotic tissue remains, it is essential to use more extensive tamponage and drainage through the pouch of Douglas and the vagina. In this procedure, several mistakes are commonly made. In an attempt to secure drainage of the pelvis through the pouch of Douglas, it is not easy to open the vagina with curved scissors and often the tissues are irregularly cut and bruised.

After supravaginal hysterectomy, it is difficult to open the posterior vaginal arch. When a long curved forceps has been introduced into the vagina, it is necessary for the operator to guide an assistant's hand in the protruding of the posterior vaginal arch by means of the instrument. After cutting through the posterior vaginal arch, we sometimes have to cope with hæmorrhages from the vaginal wound. Therefore, it is wise to place a suture at the right and left sides on the edges of the vaginal incision, although it may be difficult to do this because the edges of the vaginal incision lie deep in the pelvis.

In order to avoid some of the usual difficulties, we have made use of an instrument which consists of two movable arms held together by means of a set screw. The curved arm carries a sphere which is introduced into the vagina as far as the posterior vaginal arch shortly before the operation, after the abdominal wall and vulva have been disinfected (Figs. 1 and 2).

During the operation, the surgeon can move the handle of this instrument so as to make the posterior vaginal arch protrude into the abdominal cavity and extend upward so that it can be reached quite easily (Fig. 3). On the sphere there is a longitudinal and a transverse furrow: the former facilitates the incision in a forward or backward direction; the latter serves to pull the thread on the right or left side through the edge of the vaginal incision so that it cannot glide off the smooth surface of the sphere. The transverse

Fig. 1.

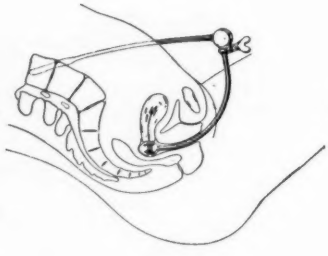

Fig. 2.

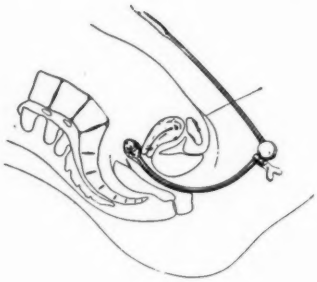

Fig. 3.

Fig. 1. Drawing of instrument and detail of spherical head.

Fig. 2. Instrument in use.

Fig. 3. The handle of the instrument has been turned so as to protrude the posterior vaginal vault into the abdominal cavity.

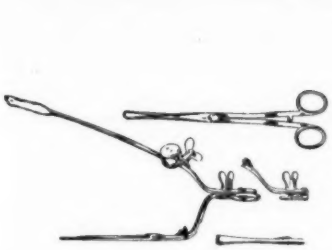

Fig. 4. Another attachment and forceps.

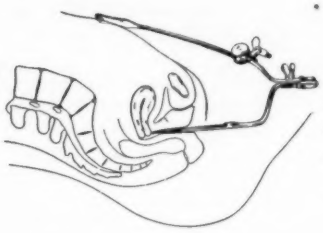

Fig. 5. Application of instrument.

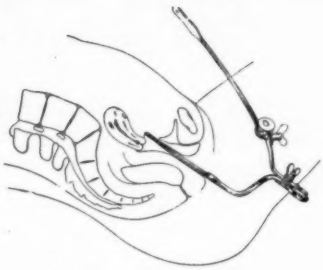

Fig. 6. Instrument in use.

furrow is also useful when a transverse incision is used in a total hysterectomy. In the foremost part of the longitudinal furrow, there is a little bridge around which can be turned the end of a drain or to which a thread may be attached that will carry the drain into the vagina when the instrument is withdrawn.

A similar attachment but provided with a crowbill and different forceps which can be hooked on to a portion of the vagina, is also very useful (Fig. 4).

Figure 5 illustrates the manner in which the forceps is fixed and the other part of the instrument is screwed upon the handle of the forceps so that, at any given moment, the operator may seize the handle of the instrument and at once bring to his hand in the abdominal cavity a well defined part of the vaginal wall (Fig. 6).

The Museaux-forceps has the advantage that when pressure is exerted on the instrument, the forceps will not pierce the tissues. In ovarian implantation, the instrument can be used to draw an ovary into the uterus by means of a thread.

The seizing of certain parts of the vaginal wall, chiefly the foremost vaginal arch, and the possibility after laparotomy of recognizing distinctly this part of the vagina and of pushing it upward, is extremely advantageous in Wertheim operations for carcinoma of the cervix and even in cæsarean operations when a drain is to be placed in front of the incision in the uterus and led downward along the vagina.

AFTER-TREATMENT OF PELVIC AND ABDOMINAL SURGICAL CASES[1]

By RALPH WORRALL, M.D., M.Ch., F.A.C.S. (Hon.), Sydney, Australia
Consulting Gynecologist to the Sydney Hospital and Surgeon to the Coast Hospital

IN the early days of pelvic and abdominal surgery, the preparation and after-treatment of the patient were considered of importance almost equal to that of the operation. Now the pendulum has swung in the opposite direction; surgeons do not hesitate to operate in the suburbs or country, and leave the after-treatment absolutely to the discretion of the local practitioner.

In a large proportion of instances, the patient does not suffer any material injury from this divided responsibility, but every once and again it happens that the absence of "the shepherd who owns the sheep," the operator himself, leads to disastrous results. I wish to cite some post-operative complications in which close "follow-up" by the surgeon averted failure and dashed death aside. The cases have been selected as illustrating different conditions and the treatment adopted.

Secondary hæmorrhage following plastic operations on the cervix and vagina is usually caused by too tight or non-absorbable sutures and needs prompt and skillful treatment. The patient should be placed across the bed, the vagina should be thoroughly irrigated with peroxide of hydrogen, 1 in 4, until all clots have been removed. Thin, narrow-bladed retractors should be passed anteriorly and posteriorly, and a firm gently-placed packing with iodoform gauze should be carried out. The foot of the bed is then raised 18 inches. If this does not control the bleeding, the patient is anæsthetized and the bleeding point exposed by the narrow-bladed retractors, and a catgut suture or sutures passed beneath it with a handled needle. In such circumstances a handled needle is much more effective than a needle on a holder. I have practiced this procedure on several occasions without interfering with the successful result of the operation.

Secondary hæmorrhage (intraperitoneal) is an untoward occurrence which I have not seen in the last 25 years. Separate ligation of vessels and abandonment of mass ligatures has abolished it.

Suppuration of an abdominal wound occasionally occurs after an operation for cancer of the cervix uteri or suppurative conditions. It may also result from the suppuration of a hæmatoma as a result of bleeding from a small vessel in the parieties which has escaped notice and ligature. It should be suspected if there is a persistent evening rise of temperature not associated with other serious symptoms. The supporting sutures being left intact, the line of incision, which has probably healed perfectly, is gently palpated, and when tenderness or a soft spot is detected, a sharp-pointed scissors is inserted, an opening made, and the pus evacuated. The pocket is rarely beneath the aponeurosis. If it is of any size a small drainage tube may be inserted and given very gentle irrigation with peroxide of hydrogen daily.

BURSTING OPEN OF AN ABDOMINAL WOUND

Case 1. After hysterectomy, the wound was closed in layers. Catgut was used for the peritoneum; kangaroo tendon, for muscle and aponeurosis; and catgut, for subcutaneous tissue and skin. The patient convalesced well. During a sudden movement on the eighth day, the wound burst open. I saw the patient within half an hour. She was etherized in bed, the dressings were removed, the abdomen was surrounded by sterile towels, the intestines were cleaned with warm saline solution and carefully returned, and the wound was again closed by catgut for peritoneum, through-and-through silkworm gut for all other structures, and separate plain catgut for aponeurosis and skin. The patient made a good recovery. There was a strong scar.

Case 2. (This occurred during the war when the house surgeons came and went every 2 or 3 weeks.) After total hysterectomy by the method which I have described in a previous article,[2] the wound had been closed by continuous plain catgut for the peritoneum, through-and-through interrupted silkworm gut sutures for all other structures, and continuous plain catgut for aponeurosis, fat, and skin; the silkworm gut sutures tied over iodoform gauze. On the seventh day, the house surgeon noticed a little serum from the lower angle of the wound, and against standing orders, removed the silkworm gut supporting sutures. Several hours later the wound burst open and the intestines extruded. I was not available so the house surgeon cleansed and returned the intestines and resutured the wound. The patient, who had previously been convalescing well and promising to be the eighty-sixth consecutive successful case treated by this method, died 4 days later of septic peritonitis. The house surgeon should have cut only the lowest silkworm gut suture and evacuated any serum or liquid fat from the lower angle of the wound.

PELVIC SUPPURATION

Case 1. Miss D. M., consulted me November 2, 1921, for gonorrhœal endocervicitis and urethritis of 2 months' standing. There had been menorrhagia for several years. On November 5, Skene's tubes were slit up and cauterized, under ether. Cervical mucosa was completely excised by a

[2] Am. J. Obst. & Gynec., 1917, lxxvi, No. 6.

[1] Presidential Address to the Section of Gynecology and Obstetrics of the New South Wales Branch of the British Medical Association.

modified Sturmdorf technique. The tubes were thickened and somewhat reddened, but fimbriated ends were not closed. Milking of the tubes failed to express any discharge. As the patient wished to avoid sterilization, the tubes were not removed. A retroversion was corrected by suturing the round ligaments behind the uterus. The highest temperature after the operation was 100 degrees, and the highest pulse was 96, but neither became quite normal and the patient felt depressed and ill. On the tenth day, 3 pints of pus were evacuated from the lower angle of the wound without benefit. On the nineteenth day, the patient began to complain of rectal tenesmus. Examination disclosed a small collection in the pouch of Douglas. Under ether, the posterior fornix was incised; about 3 drams of pus was evacuated and a split rubber tube inserted. Rapid recovery followed. The patient was seen again on September 19, 1926, that is, nearly 5 years after the operation. She had been fairly well except for nervousness which was attributed to excessive menses. There had been no dysmenorrhœa or abnormal discharge since operation. Examination showed a normal cervix. The appendages were slightly enlarged and slightly tender. The patient had been living with the same lover ever since but a condom was always used in coitus.

This case demonstrates how treacherous a gonorrhœal infection always is and what a risk is taken when an attempt is made to preserve the tubes under such circumstances. It is probable that she would not conceive even if the use of condoms were discontinued A total hysterectomy would have left her in better health, provided the ovaries were not removed

Case 2. Mrs. G. B., 34 years of age, nullipara, consulted me on January 9, 1924, for pain in the lower abdomen, rectal tenesmus, and dysuria which had troubled her for 2 days. There had been 3 similar attacks preceding the last 3 periods. Temperature was 101.4 degrees; pulse, 100. Vaginal and rectal examination disclosed a myomatous uterus and a tender fixed mass in Douglas's pouch. The diagnosis was inflamed or suppurating ovarian cyst complicating myoma.

On January 11, under ether, posterior vaginal cœliotomy was done and the supposed cyst was carefully separated. The abdomen was then opened and subtotal hysterectomy was performed, both appendages which were normal being left. The supposed cyst proved to be an enlargement of the pelvic colon, hard and nodular, resembling a malignant growth. The bowel was divided well above and below the growth and after thorough removal of the latter with the surrounding tissue, the bowel ends were re-united by the Rutherford Morrison method—passing a large tube down through the anus, suturing the upper end inside the proximal bowel, making traction on the lower end, and invaginating the upper end of the bowel into the lower, then suturing with fine chromic gut. A small rubber and gauze drain was passed into the vagina through the vaginal cœliotomy wound.

The bowels moved by castor oil in 50 hours. The bowel tube and drain were removed on the eighth day. Temperature and pulse continued as before the operation. On the eleventh day, a small quantity of pus was evacuated from the lower angle of the abdominal wound and there was a temporary drop in temperature. On the seventeenth day, a rigor took place and the temperature rose to 101.4 degrees; pulse, 104. Micturition was frequent and painful and the urine showed a trace of pus. Vaginal examination disclosed a slightly tender exudate in the anterior fornix.

Under ether, the bladder was dissected up and the finger pushed into a collection of pus in the uterovesical pouch. A split rubber tube was inserted here, and another in the old opening in the posterior fornix, dilated for the purpose. After this there was an immediate improvement in all the symptoms. The rubber tubes were removed on the fourth day, and the patient left the hospital 6 weeks after the original operation, feeling very well.

Professor Welch reported the specimen to be one of diverticulitis. "One of the most remarkable I have ever seen: there are only two diverticula—one extends beneath the peritoneum and is on the point of perforation, the other has also penetrated beyond the muscular coat, into the mesentery where a small necrotic abscess has developed."

When, as in this case, it is not possible to lift the bowel out of the pelvis so that it can be segregated prior to resection, it is difficult to guard against infection and suppuration. On March 31, 1925, 15 months after operation, the patient reported as having "had no trouble since." Assiduous follow-up had saved this life.

POSTOPERATIVE INTESTINAL OBSTRUCTION

Vigilant watch should be kept for the dangerous complication of postoperative intestinal obstruction. Promptness is the very essence of its successful treatment. If vomiting associated with paroxysmal pain occurs in a patient who has been doing well for a week or so, and if two large enemata fail to bring away flatus and fæces with relief from the symptoms within 12 hours, a diagnosis of intestinal obstruction should be assumed, and appropriate measures taken without delay. In the following case, and another exactly similar to it, I adopted a method of treatment which is not mentioned in any of the well known textbooks and which in these cases was attended by success. The method is worth bearing in mind, but should be used with discrimination. The surgeon must be in a position to exclude definitely peritonitis or suppuration.

Case 1. Mrs. Z., age 44 years, was admitted to the Lister Hospital, January, 1923, suffering from hæmorrhage due to myomata. An operation had been performed by another surgeon several years previously for retroversion and salpingitis. I did a total hysterectomy leaving both ovaries (the tubes had been removed at the previous operation). Moderate adhesions were present. The patient convalesced easily until the sixth evening when she was seized with paroxysmal pain and vomited several times. Two large turpentine enemata during the night failed to move the bowels or bring away flatus. When I saw her the next morning she looked anxious but not very ill. Pulse was 100; temperature, 99.6 degrees. The abdomen was moderately distended but not tender. Vaginal and rectal examinations disclosed nothing abnormal.

While preparations were being made for the reopening of the abdomen, I thought I would try succussion. Standing on the bed at the foot, I seized the patient's legs beneath the knees and lifting up her body so that she rested on the back of her neck, and aided by the spring mattress, I gave her three fairly vigorous shakes. Another large turpentine and soapy water enema was then administered. This acted promptly and brought away fæcal matter and much flatus. The subsequent convalescence was uneventful.

CASE 2. E. P., 50 years of age, iv-para, had suffered from hæmorrhage for a year. On October 26, 1916, a total hysterectomy was performed. The operation was difficult because of the obesity of the patient and the depth of the pelvis. The patient convalesced well until the thirteenth day when she vomited several times. The bowels moved the next day but vomiting continued. I did not see the patient until the following day. She was then moribund and died 5 hours later. Postmortem examination showed the ileum greatly distended and adherent to the cervico-vaginal angle on the left, about 18 inches from the cæcum. The ilium proximal to the adherent point had undergone axial rotation, hence the obstruction. The adhering bowel was easily separated. I wrote on the report of the post-mortem, "C'est dommage." Indeed, it was a pity that owing to the War and the dislocation of the interne service of the hospital, steps were not taken to relieve the obstruction on the day when the vomiting started. I regret that even at that late hour I did not give this poor woman the chance of an enterostomy performed under local anæsthesia.

CASE 3. Strangulation of the ileum by an omental band, with recovery after division of the band and lifting of the small bowel out of the pelvis. A right ovarian cyst had been removed 18 years previously during pregnancy and without interruption of the same. The patient complained of pain in the lower abdomen, nausea, and loss of flesh for 2 or 3 years. On April 29, 1920, I removed a large pseudo-mucinous ovarian cyst on the left. Some of the contents of a thin loculus escaped on the pads. Omental, small intestine, and sigmoid adhesions were present. The appendix was also removed.

The patient convalesced well until the ninth day when she vomited twice. The bowels which had been acting regularly, could not be moved on the tenth day, notwithstanding the administration of three enemata. No flatus could be brought away. The temperature rose from normal to 99.2 degrees; and the pulse to 104. The abdomen was fairly flat until the tenth day when slight distention appeared. The patient said she could feel the wind going to a certain point where it stopped. The abdomen was re-opened at 8.00 p.m. that same day by incision at the outer border of the left rectus. The ileum and an omental tag were found to be adherent in Douglas's pouch. On separating and lifting up these structures, we found the ileum, which was of a deep, claret color, but still shiny, strangulated by the omental tag. The bowel was dilated above and collapsed below the point of constriction. The bowel contents were easily moved on. The omentum was re-arranged and the tag removed.

The patient passed flatus immediately on being returned to bed, while a rectal saline was being administered. The bowels were moved the next morning by an enema, preceded by an intramuscular injection of pituitrin. The pulse which had been 112 after the operation was 96 the next morning. No further vomiting took place. Convalescence subsequently was easy and both wounds healed perfectly.

Case 4. Intestinal obstruction successfully treated by enterostomy. Mrs. F. B., 44 years of age, was examined on June 5, 1924. Seven years previously, she had had puerperal sepsis following labor. Hæmorrhage had continued for a year after that. A repair operation had been performed and the tubes had been ligatured so as to prevent further pregnancies. Menses continued to be very excessive. The patient had taken brandy for weakness. She was anæmic and stout. The uterus was bulky; the cervix was lacerated and degenerated; the right appendages were enlarged. At operation on June 28, great and dense adhesions of the omentum were found. The right ovary had been transformed into a cyst. Total hysterectomy with removal of the right ovarian cyst was carried out. No sign of ligatured tubes was evident. The appendix was in the 11 o'clock position, and was not removed. A large amount of granulation tissue was found in the uterine cavity which the pathologist reported as "non-malignant."

Convalescence was easy until the eighth day when the patient complained of pain in the lower abdomen and vomited twice. Two enemata failed to move the bowels. The lower abdomen was distended and slightly tender. The same evening, under ether the lower part of the wound was re-opened, but when the finger was pushed down only a little blood was evacuated. A split rubber drain was passed into the vagina through the shell of the cervix. A gridiron incision was then made in the right iliac region, a distended coil of ileum presented. This was incised through a pursestring suture, a drainage tube was inserted into the bowel and sutured with one suture of fine catgut. The first pursestring suture was then tied tightly encircling the rubber tube. Another pursestring was passed and tied in the same way. The bowel was not sutured to the parieties but the tube was sutured to the skin to prevent an accidental pulling out. The distal end of the tube was inserted into a baby's feeding bottle which was tied to the patient's side. Large quantities of very foul dark fluid escaped and when the flow slackened the bowel was irrigated through the tube with normal saline. Vomiting ceased at once; the bowels moved after an enema in 48 hours. The tube was allowed to come out on the tenth day and the tract had practically closed 15 days later. The temperature kept at 102 or 100 degrees and the pulse was 112 to 100 for 8 days. On the sixteenth day both rose again to 100 when slight left femoral phlebitis showed itself. The patient was discharged well 48 days after the first operation. The patient was seen again on February 5, 1926, and was found to be free of any serious trouble. Bimanual examination disclosed nothing abnormal.

Case 5. Intestinal obstruction treated first by multiple incisions and two days afterward by enterostomy. Mrs. W. E., 40 years of age, was seen on March 22, 1920. Operation for retroversion had been performed 6 years previously. She complained of excessive menstruation, weakness, and anæmia. Operation disclosed a moderately adherent omentum beneath the scar. The left ovary which was a cyst with tarry, bloody contents, and was adherent to the pelvic colon, was removed. The uterus was much enlarged, riddled with myomata; the cervix showed marked cystic degeneration. A total hysterectomy was done. A diverticulum from the cæcum which was adherent to the right fallopian tube was removed and the proximal end was buried. The appendix was normal and not removed.

The patient convalesced fairly well. The pulse was never quick but was weak. There was vomiting and distention for the first 2 days, but it was relieved by enemata. The patient progressed favorably until the night of the sixth day when vomiting returned and became fæcal. The temperature remained normal and the pulse was only 86. At 10 o'clock the next morning, the soundly healed wound was opened and a slight amount of turbid fluid escaped. The small intestines were distended and reddened. A loop adherent in Douglas's pouch was markedly kinked and partly collapsed beyond the kink, some 16 inches from the cæcum. The distended bowel was incised in 3 places and very foul-smelling, dark fluid was evacuated. The opening was closed with a fine chromic gut. The pulse was 112 after operation. The bowels moved well that evening by turpentine enema, preceded by intramuscular injection of pituitrin.

On the seventh day, the pulse was 104 The abdomen was still slightly distended. No vomiting after 1 p.m. the preceding day. On the afternoon of the eighth

day, the vomiting returned; distention increased; and the pulse rose to 120 and was very weak. The patient's arms were quite cold, and she seemed moribund when I saw her at 8 p.m. Ether was given and an incision was made in the right iliac region. A distended loop of ilium close to the cæcum was pulled out and a rubber tube was inserted into its lumen by the technique described in a previous case. The patient was quickly returned to bed, no worse than before the short operation.

On the next day, the ninth, the patient was distinctly better. Much flatus and liquid, foul-smelling fæces were evacuated through the tube. Vomiting ceased. On the tenth day the bowels were loose; the pulse was 104; the patient was in good condition but felt "low." On April 6, the fourteenth day after the first operation and the seventh day after the insertion of the tube into the bowel, the tube was removed. The median wound was suppurating; the patient looked dull and poisoned; pulse was only 100; and the temperature was normal. After this, progress was steady and on May 29, 65 days after the first operation, the patient was discharged feeling well. The right iliac wound had suppurated and was not quite healed. A fine probe could be passed into the bowel. Six weeks later, the patient reported: "wound healed and felt well." On July 22, 1922, 2½ years later, the patient consulted me for an attack of epistaxis. She was otherwise very well. There was a slight hernia in the lower end of the median scar. The pelvis felt normal except for the absence of the uterus. I think it would have been better at the second operation to have done an enterostomy and have left in a tube instead of attempting to empty the bowel by puncturing and suturing the openings.

In the foregoing histories the symptomatology of postoperative intestinal obstruction is well exhibited. The patient does well for a few days or weeks and then suddenly vomits and complains of paroxysmal pains. Flatus ceases to pass and the bowels cannot be properly moved. The temperature and pulse remain unaffected for a time. There is increasing abdominal distention but no great amount of tenderness.

The patient's life depends upon unhesitating action. If two enemata fail to bring relief, succussion as described should be tried. If this is unsuccessful, the abdomen should be opened, the hand passed into the pelvis, and all small intestines lifted up and inspected. If the contents of the distended bowel can be easily milked into the collapsed bowel, probably all will be well; if not, an enterostomy should be done. Puncturing of the distended bowel in several places involves too great a risk of infection. I have not tried Handley's ileocolostomy with cæcostomy or Bonney's jejunostomy. The passing of the enterostomy tube through the omentum as advised by Dr. Wesley Long is a device worth bearing in mind.

I am inclined to think that many cases of paralytic ileus with peritonitis may own a mechanical basis or be associated with a mechanical obstruction and that a timely enterostomy might have saved some of my much regretted fatalities. It is now generally recognized that in death from peritonitis, the patient is poisoned by absorption of the toxins formed in the stagnant intestinal content. This suggests enterostomy as the most hopeful treatment.

ACUTE DILATATION OF THE STOMACH

Case 1. Mrs. J. M., 60 years old, was examined on October 16, 1919, and complained of attacks of gastritis, accompanied by vomiting, for 3 years. Morphia had been necessary to relieve the pain. Vomiting did not relieve the pain, but food seemed to determine an attack. The patient had lost flesh and felt weak. X-ray examination report suggested gall stones with gall bladder adherent to transverse colon and pyloris or ulcer of the pyloroduodenal segment.

On October 22, 1919, cholecystostomy was performed and 14 small, sharp-edged gall stones were removed. A tube was sutured into the gall bladder without attaching it to the parieties. Neighboring organs were normal, except for the liver which appeared "nutmeg." The patient convalesced well. The tube came away on the tenth day. There was free flow of bile from the sinus. The stools showed normal bile appearance. On the thirteenth day, the patient suddenly began to vomit large quantities, forcibly at first, later the fluid seemed to simply overflow from the mouth. There was no pain, no tenderness, and no pyrexia. The urine contained no bile. The bowels moved easily by enemata. Distention was present in the upper abdomen but not in the lower. The pulse was 100 to 112. Bile flowed freely from the sinus.

The patient was placed in the prone position, the foot of the bed was raised 18 inches. Pituitrin, strychnine, and digitalis were administered intramuscularly and the stomach was washed out several times daily. The vomiting gradually abated but continued more or less for 10 days and recurred occasionally for another 14 days. The patient was quite well 6 weeks from the day of operation.

Case 2. Mrs. A. A., 44 years old, complained of "nasty discharge, profuse menses, and great pain with periods." Examination showed a very large sessile polyp distending the vagina. Several myomata were felt in the hypogastrium. On June 17, 1924, the polyp was cut away by morcellement. Abdominal section was made and total hysterectomy performed. There was considerable subserous development. The bladder was drawn up over the face of the tumor. That afternoon the pulse was 84 and the temperature, 99.2 degrees. Partial incontinence of urine lasted for 48 hours after operation.

On June 20, the patient was still vomiting but the bowels acted well after enema. The pulse was 84 and the temperature, 99 degrees. Lavage of the stomach gave relief for 12 hours then vomiting resumed. Lavage was repeated and again the vomiting ceased. On June 22, the patient enjoyed a breakfast of egg and toast, but at 2:00 p.m. there was a sudden attack of epigastric pain and vomiting, followed by collapse. The pulse ran up to 160 and was thready. The stomach tube evacuated over 4 pints of darkish fluid and immediate improvement followed, helped, I think, by hypodermics of eserine, strychnine, and digitalone; the raising of the foot of the bed 18 inches; the keeping of the patient in a prone position; and the administration of rectal saline every 4 hours. The vomiting ceased after this. The stomach tube found no fluid in the stomach. Next day the pulse was 118 and had good volume. No further vomiting occurred until the tenth day when it returned and was again controlled by lavage. It returned again on the thirteenth day and the tube evacuated over a quart of dark fluid with explosion of gas.

There was no further vomiting after this. The supporting silkworm gut sutures were removed on the eighteenth day. The patient was apparently normal in every way and the wound perfectly united.

On the evening of the eighteenth day, the patient felt something give way when she was moving about in bed, and the intestines were found beneath the dressing. I arrived in 20 minutes and without moving the patient from her bed, or without giving an anæsthetic (¼ grain of morphine had previously been given), I washed the intestines with saline solution, replaced them, brought the omentum down, and resutured the wound as in the first instance, viz., peritoneal suture of plain gut; through-and-through to the peritoneum of silkworm gut; anterior aponeurosis, subcutaneous tissues, and skin, plain gut. The patient seemed unaffected by the untoward occurrence. The supporting sutures were removed in 20 days and the wound was soundly healed without any indication that it had burst open. The accident appeared to have been caused by the vomiting for 13 days and the great debility thereby induced.

It is to be noted that in one case, the symptoms of dilatation commenced on the thirteenth day, and in the other, on the fourth day. The diagnosis is not difficult, the bowels are unaffected, while the sudden onset, epigastric pain, collapse, and effortless vomiting of large quantities, form an unmistakable clinical picture. The only effectual treatment is as described in the case histories.

In considering postoperative complications, prophylaxis must not be forgotten. Perfect hæmostasis; careful operating; the use of quick methods and not quick movements; and asepsis will minimize the occurrence of these untoward events.

One word on a different aspect of the subject. In the treatment of all disease, we are fighting a losing hazard. Sooner or later death will triumph and "will seize the Doctor too." In that hour, the dominant note should be infinite compassion. This should prevail over all other considerations; morphine must not be spared. In the words of Dr. Robert Hutchinson: "When our time comes, let us hope we may fall into the hands of a doctor who knows the value of morphia."

THE SURGICAL IMPORTANCE OF X-RAY EXAMINATION OF THE ŒSOPHAGUS AND THE PHARYNX

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THE ŒSOPHAGUS

THE diagnosis of diseases of the œsophagus has progressed surprisingly since the dawn of the roentgen era. Up to that time, the œsophagus could be examined only by means of sounds, a method which must be looked upon as not altogether harmless for the patient (aneurisms, diverticula), quite apart from its inconvenience and frequently faulty results. At about the time X-ray diagnosis appeared, medical diagnosis was also enriched by the addition of the method of œsophagoscopy, the importance of which is generally recognized in the surgery of foreign bodies of the œsophagus and extractions in general. This method has served us well in the search for important findings in pathological conditions of the œsophagus.

X-ray examination of the œsophagus is painless, harmless, convenient, and very valuable. The method depends, as is well known, on the fact that the contrasting agent, the paste, is swallowed *while the patient is standing upright* and is held for a longer or shorter period of time at the level of the stenosis (we will consider stenoses almost exclusively in this paper). This affords us knowledge as to the *upper level* of the stricture. We can also make important conclusions as to the nature of the process on the basis of the type of the picture. The results obtained by a fluoroscopy in upright position (these can be completed by a plate) are nevertheless lacking in certain important diagnostic facts. Such an examination gives us a good view of the upper border of the process, but it does not inform us as to the *extent of the condition in the œsophagus*, since the contrasting mixture passes through the œsophagus in a regularly narrow line. In order to fill this great need in œsophagus diagnosis, our clinic pointed out (6 and 7) the importance of X-ray examination of the œsophagus in cases of stricture in horizontal position of the patient. The patient swallows the contrasting mixture *after taking up a horizontal position*. The transportation of the mixture through the incompletely stenosed œsophagus is a slow one in this horizontal position and the result is a complete filling of the œsophagus. In incomplete stenoses, this means not only a cast of the contours over the stricture, but also within and below the area of stenoses. Although this method of examination is of little value in tumors of the thoracic section of the œsophagus because of the dubious prognosis of operation in such cases, it is all the more important in cicatricial strictures. An X-ray examination of an incomplete cicatricial stricture of the œsophagus in this way gives us an idea as to the upper level of the stricture, its length, and form. In short, we are no longer in the dark but are now in a position to learn whether a stenosis is extensive or small, and this gives us a better foundation for the endoscopic treatment of the scar.

In examining the patient in a horizontal position, it is better to irradiate from a purely lateral position than from an oblique one, as we have previously mentioned (4) for the examination of the trachea following *Lilienfeld's* suggestion for the lateral photography of the sternum.

The patient lies completely on one side (1) at an angle of 90 degrees to the table. The shoulders are drawn firmly backward in order to remove the almost impermeable humeral epiphyses from the field of irradiation; the arms are drawn back and the hands clasped firmly together. In this position, with the humeral epiphyses directly over each other and the knees drawn up, the patient is fixed on his side. The head is supported by means of sandbags, so that it is in normal position. The plates are 24 by 30 centimeters. The central ray falls on the midpoint of the distal clavicle and strikes the midpoint of the plate in the line of its projection (this is for the examination of the cervical section and the proximal portion of the thoracic section of the œsophagus). In order to examine the distal portions of the œsophagus, the plate and the tube must be displaced a suitable distance distally, and the focus must be correspondingly lower.

While lying in horizontal position the patient swallows a contrasting mixture of medium consistency. Before a plate is made, the patient is examined on the trochoscope under fluoroscope in horizontal side position. At this time, one can observe how long it takes from the time the paste is swallowed until the œsophageal defect is completely filled. After the preliminary fluoroscopy, the plate is made with the Bucky-Potter diaphragm while the patient is swallowing more of the contrasting mixture. The plate should be

taken at a moment after the swallowing act has begun, the exact time having been judged during the swallowing at the preliminary examination (the interval between the beginning of swallowing and the snapping of the plate is between a quarter and a whole minute). It may appear more practical to some to combine the trochoscopic examination and the plate, by snapping the latter during a satisfactory moment. This, of course, dispenses with the Bucky-Potter diaphragm.

Figure 1 illustrates the value of X-ray examination of œsophagus tumors in horizontal position. The lateral picture shows the location, size, and topographical relationships of the tumor; the degree and the length of the stricture, as well as the shape of the œsophagus above and below the stenosis, are also clearly depicted. We definitely see not only the upper, but also the lower border of the stricture. It is often possible in the lateral projection, as in this case, to portray the tumor itself which stands out against the less dense lungs. Therefore, this is definitely an œsophageal tumor which is localized in the proximal part of the thoracic section, causing an incomplete stenosis of the tube. The stenosis begins somewhat below the level of the upper thoracic aperture and reaches down to about the level of the bifurcation. The thoracic section of the trachea is pushed forward and compressed from behind.

As we have already mentioned, the information we obtain on the form of incomplete cicatricial strictures of the œsophagus by means of lateral irradiation is much more important than our results in the study of the tumors. Examination in upright position gives us information only on the upper level of the stenosis without orienting us on the lower portions of the œsophagus. In the event that the stenosis involves but a small portion of the tube, it is important to know this. We must also ask ourselves whether the stenosis is extensive or not, what the shape of the stricture is, and at what points passage is still possible. These questions must be definitely answered before any purposeful endoscopic treatment can be undertaken. The better we know the condition present, the better will be our results in treatment.

The advantages of examining the patient in the horizontal position as against the upright position are illustrated in Figures 2 and 3. They demonstrate scarry strictures of the proximal portions of the œsophagus following hydroxide cautery. In Figure 2 (examination in upright position) the contrasting mixture is seen to collect at a point above the bifurcation; below this point there is a thread-like filling down to the cardia. The picture gives no information as to whether the whole œsophagus down to the cardia is stenosed or whether only a small section of the tube is affected. Figure 3 (examination of the same patient in horizontal position; the contrasting fluid swallowed in horizontal position) shows that the stricture is only very short, that the œsophagus widens normally 2 centimeters below this point. The stricture is due to a cicatricial shrinkage which has drawn the anterior wall of the œsophagus toward the back. A small passage, corresponding to the posterior wall of the tube, has been left. We are thus able to learn the length of the stricture, its form, and the point at which a passage has been preserved (in this case along the posterior wall). Two centimeters below the upper level of the stenosis there is again a normal breadth to the lumen. The greater width of the œsophagus above the stricture is the result of a compensatory widening above the stenosis.

Figure 4 shows how well the X-ray examination of the patient in horizontal position can reveal finer stenoses of the œsophagus (scar stricture following lye cautery). Despite the unusual narrowness of the œsophagus in this case, there is seen a complete filling of the tube not only above, but also below the stricture; so that we have a complete picture of the cicatricial shrinkage in the œsophagus. It is natural that only incomplete stenoses of the œsophagus are to be considered for examination in this way. For if the stenosis were complete, its form could be definitely cleared up only by a simultaneous filling from above and an œsophagoscopy from below through a gastrotomy. Sometimes we can also get a general idea as to the length of a scarry stenosis by examining in horizontal position without filling the œsophagus. The lateral view of the cicatricial tissue discloses the latter to be denser than the air filled lungs.

We must also mention the case of a *congenital, partial œsophagus stenosis*. The patient was a 12-year-old boy who had always suffered from marked swallowing complaints. X-ray examination with the patient in upright position (Fig. 5) showed the transient collection of the contrasting mass in the œsophagus at about the height of the bifurcation. The œsophagus was rather markedly dilated above this point, but from this level downward the œsophagus was seen only as a thin thread. Here again we were unable to decide whether the stricture reached as far as the cardia or not. Figure 6 was made with the patient in the horizontal position (the patient swallowed the paste in this position). It shows that the stenosis is 6½ centimeters long and that below the stricture the œsophagus is again normally dilated (at *A*). But this examination disclosed still another interesting point, and

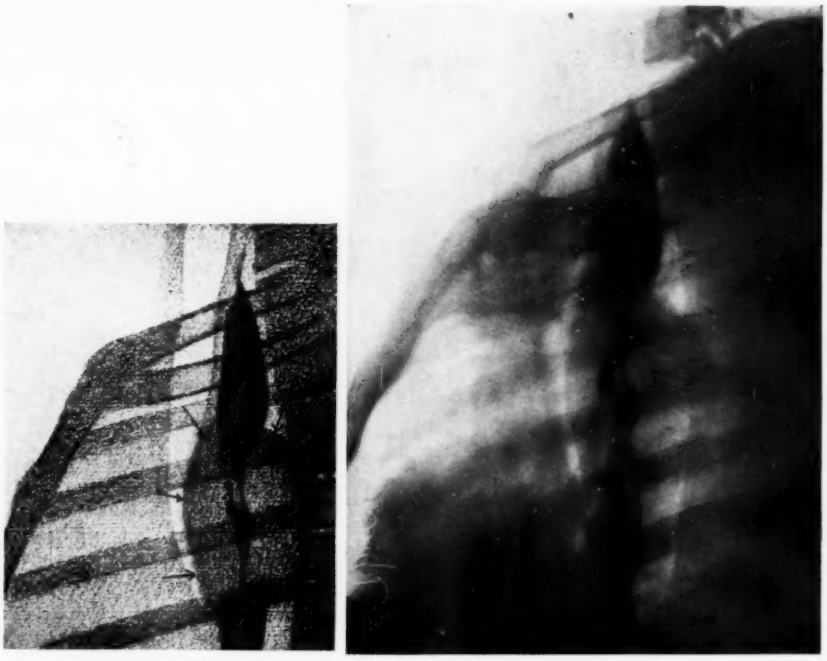

Fig. 1. Œsophageal tumor examined with the patient in horizontal position. The patient swallowed the paste in this position. The original photograph is at the right; the sketch is at the left. One can see the upper and lower poles of the stricture and the form and extent of the stenosis. The tumor itself is clearly visible in this projection (marked by the arrows). The thoracic portion of the trachea is compressed from behind.

that is the presence of a second stenosis of the œsophagus somewhat below the height of the upper border of the sternum (at *B*). This stenosis was hardly visible in the examination in upright position.

X-ray examination of the œsophagus in horizontal position is not only valuable for the study of strictures, but also in the diagnosis of pathological dilatations, especially since bulgings of the œsophageal wall, as Kloiber (2) has already mentioned, fill themselves much better with the contrasting paste in horizontal position than in the upright one. The double Figure 7 illustrates these conditions and demonstrates a large *pressure diverticulum* at the height of the cricoid cartilage which, as is visible in lateral view, takes its origin at the posterior wall of the œsophagus in the cervical portion of the latter. The œsophagus has been pushed forward by the diverticulum and has also been quite badly compressed from behind.

The X-ray examination in horizontal position is, however, more important for the diagnosis of *traction diverticula* than that of pressure diverticula. The former are not infrequently revealed only by this method of examination, since a more complete filling of the œsophagus is attained in the method as a result of the slower passage of the contrasting paste (this was the reason why *Palugyay* (3) recommended the examination of patients with traction diverticula in raised pelvis position). We remember a few cases of middle aged persons with swallowing complaints in whom we suspected carcinoma of the œsophagus. X-ray examination in horizontal position showed that the cause of the stenosis was an acute angled bulging of the œsophageal wall, a traction diverticulum. These traction diverticula can sometimes even lead to severe symptoms of stenosis, as we were able to observe in a middle aged woman who also had a kyphoscoliosis resulting from a vertebral caries in the earliest years of life. The patient suffered from frequent and increasing complaints in swallowing, which finally reduced her to a liquid diet. X-ray examination in upright posi-

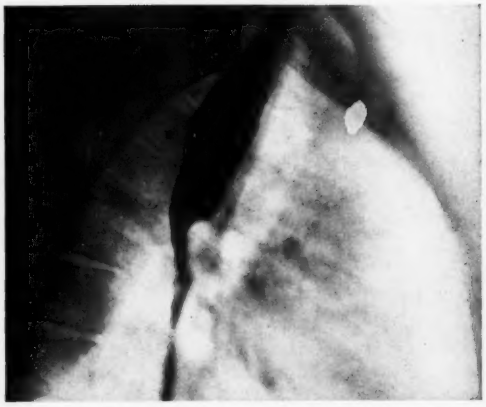

Fig. 2. Cicatricial œsophageal stricture above the level of the bifurcation (lateral projection). The examination was made in upright position. The length of the scarry stenosis is not recognizable.

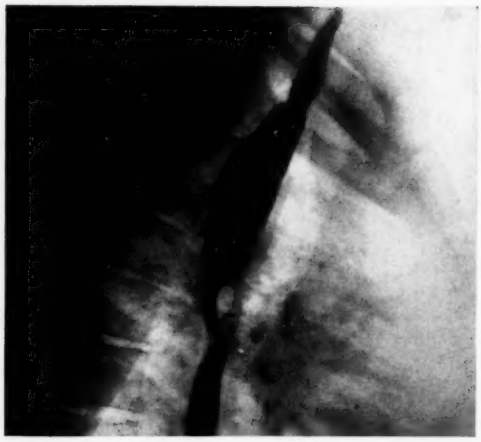

Fig. 3. Cicatricial œsophageal stricture above the level of the bifurcation (lateral projection). The same patient as in Figure 2. Examination was made in horizontal position. The length of the scarry stricture is easily recognizable.

tion disclosed but *one* angulation of the wall in which the contrasting fluid collected for some time; but examination with the patient in horizontal position revealed several distentions on the anterior and posterior walls of the œsophagus (traction diverticula). The patient underwent gastrotomy later, when the complaints increased.

Fig. 4. Unusually narrow cicatricial stricture of the œsophagus at the middle of the thoracic portion. The examination was made in horizontal side position. There is a complete filling of the œsophagus within and below the stenosis despite the exceptional narrowness of the latter. This makes the form of the scarry lesion easily recognizable.

THE PHARYNX

X-ray examination of the pharynx meets with great difficulties as the contrasting fluid swallowed with patient in upright position practically never gives a satisfactory cast of the pharynx, but rather resolves into drops and crumbs because of the breadth and peculiar form of this formation, instead of passing in a solid column as it does in the œsophagus. Another deterring circumstance is the fact that the act of swallowing is so quick, that a detailed fluoroscopic examination is impossible. We refer, of course, to only the mesopharynx and hypopharynx, since the ingested food passes only these sections. These portions of the pharynx lie between the soft palate and the pharyngo-epiglottic folds on the one side (mesopharynx), and the pharyngo-epiglottic folds and the region situated behind the lower border of the cricoid cartilage, on the other (hypopharynx). X-ray examination of the epipharynx which corresponds to the region of the upper choanæ, is even less fruitful. In the following paragraphs we wish to describe what is possible in the way of X-ray examination of tumors of the mesopharynx and hypopharynx.

As we have already mentioned, examination in upright position cannot show us much in these portions of the tract. Only a very marked hin-

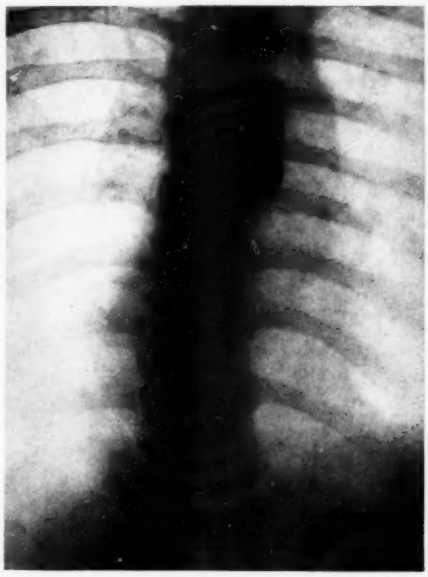

Fig. 5. Congenital stenosis of the œsophagus in a 12-year-old boy. Examination was made in standing position. There is transient retardation of the contrast mixture at about the level of the bifurcation. The œsophagus is rather dilated above this point, and below it, the tube is visible only as a narrow thread. The length of the stricture is not recognizable.

drance can be recognized at this point by means of X-rays. Of course, even then we will be able to see only the upper border of the stenosis, which is not of much value, since the laryngologist can learn this much more conveniently and quickly by the use of his mirror. It is much more important to determine the extent of a tumor than the fact of its presence in this case, especially since the laryngologist is more or less limited here. In the case of a tumor which projects into the lumen of the pharynx, the laryngologist can certainly see the upper pole of the growth, but he can tell us nothing of the length of this growth because the prominence of the tumor precludes his looking into the tube. These are precisely the cases which are of such great importance, since the operability and the indications for further treatment—operative or conservative—depend upon just such conditions. Even Hajek recommended anterior mediastinotomy in such cases for diagnostic purposes, so that one might become oriented on the extent of the tumor. If, therefore, X-ray examination of these conditions can reveal anything, it will solve a real diagnostic problem.

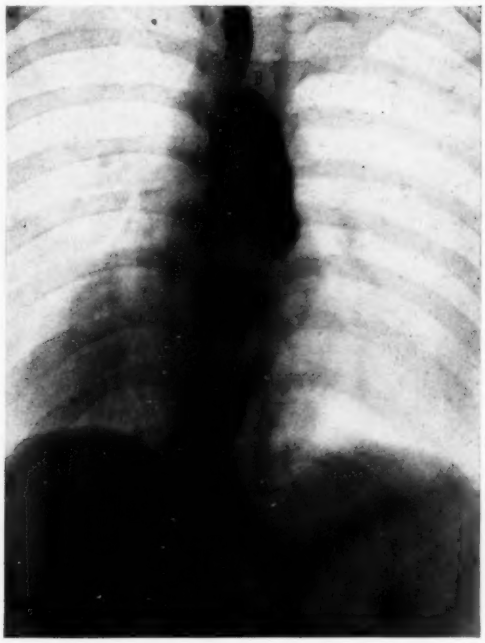

Fig. 6. Congenital stenosis of the œsophagus in the same patient as in Figure 5. Examination was made in horizontal position. It is clear that the stenosis is 6½ centimeters long and that the œsophagus becomes normally dilated again below the stricture (at *A*). There is also a second stenosis somewhat below the level of the proximal sternal margin.

In order to reach some definite conclusion, we undertook the examination of pharyngeal tumors with the same method we have used in œsophageal strictures, i.e., with the patient in horizontal position. The contrasting paste was also given in horizontal position (5). The passage of the paste is slower in the œsophagus than in the pharynx. The radiological analysis of the swallowing act is thus easier than it was formerly. As a result of this retardation of the swallowing act, the mass of a large bite of the paste fills the mesopharynx and hypopharynx rather well, but even more so under pathological conditions in which the impulse to swallow is lowered.

The patient lies on the trochoscope during the examination on one side, the shoulders are pulled backward and the head is supported by sandbags. The backward pull on the shoulders also permits a view of the œsophagus, which is of importance in that pharyngeal tumors often affect the œsophagus. Lateral projection simultaneously shows us a

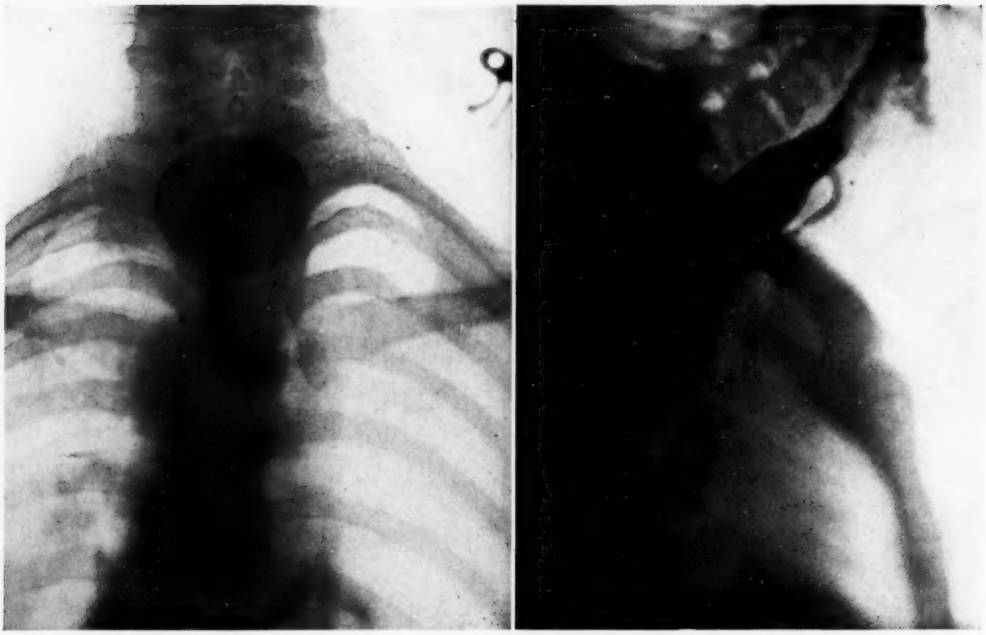

Fig. 7. Examination was made in horizontal position. At the left is the anterioposterior projection. At the right is the lateral projection. A large pressure diverticulum is to be seen which arises from the posterior wall of the œsophagus. The lateral projection very clearly shows the filled diverticulum over-flowing.

clear picture of the larynx and this is an aid in orientation. After fluoroscopy, a careful plate is made, or the X-ray plate can be made separately with the Bucky-Potter diaphragm a few seconds after the paste has been swallowed. In the latter case, it may be necessary to take the plate several times in succession in order to catch the complete filling of the pharynx.

The following illustrations demonstrate what important information such an examination can give. Figure 8 shows the filling in a normal mesopharynx. More than half the swallowed paste has already passed the epiglottis. The picture has a regular and sharply defined contour. Orientation is further simplified by the laryngeal picture which is also visible in the plate. One can see the free portion of the epiglottis, its pedicle, and its continuation into the ligamentum thyreo-epiglotticum from the lower border of which the vocal cords pass to the plainly visible arytenoid cartilages. Two fingerbreadths lower, one sees the lower border of the cricoid cartilage, the tracheal aperture, simultaneously the lower margin of the hypopharynx, and its transition into the œsophagus.

Figure 9 illustrates a pathological case, a tumor of the mesopharynx which protrudes into the hypopharynx and even farther into the cervical portion of the œsophagus. As one can easily see, the upper pole of the tumor lies just below the level of the hyoid bone. At this point a greater quantity of the contrast fluid has collected like a cupola over the upper pole of the tumor. The tumor affects the posterior and lateral portions of the mesopharynx and hypopharynx and has left but a small passage for the paste at the anterior wall of the pharynx. The picture also reveals the characteristically irregular and fringed border of tumors. The posterior wall of the pharynx is lifted from the spinal column because of a marked swelling of the former, this simultaneously seeming to push the cervical part of the trachea forward. There is no transgression of the tumor to the spinal column—a point which is very important in respect of the operability of such cases. It is nevertheless impossible to operate in this case because of the great extent of the tumor, especially since the neoplasm has already begun to affect the cervical portion of the œsophagus. A normal filling of the œsophagus is to be seen only

Fig. 8.

Fig. 9.

Fig. 10.

Fig. 8. Partial cast of a normal mesopharynx.

Fig. 9. Tumor of the mesopharynx which penetrates to the hypopharynx and also extends toward the cervical portion of the œsophagus. The upper pole of the tumor lies in the region just below the level of the hyoid bone; the lower pole is just above the level of the upper thoracic aperture.

Fig. 10. Pharyngeal tumor. The upper pole is seen at the level of the arytenoid cartilages, the lower pole being at the level of the clearly visible calcified cricoid cartilage.

just above the level of the upper thoracic aperture. The lower pole of the tumor is probably to be found at this point.

Figure 10 illustrates a tumor of much smaller size. In this case the upper pole is easily recognized at the level of the arytenoid cartilages; the lower pole at the level of the clearly visible, calcified plate of the cricoid cartilage. The tumor was removed by Hajek.

Figure 11 shows a tumor of the hypopharynx. The upper and lower poles of the tumor are easily recognizable, as are also the length and form of the stricture. The tumor is proliferating toward the larynx and forward toward the cervical portion of the trachea; it has already pushed the latter forward and compressed its posterior wall.

If the tumor is large, it is often difficult to get the upper and lower poles on the plate at the same time. In such cases we make one picture of the upper pole with a part of the stricture; and in a second picture we can then catch the lower pole with the stricture above it during a later phase of the act of swallowing. Such a double picture is shown in Figure 12. The picture on the left shows

Fig. 11. Tumor of the hypopharynx. The upper and lower poles of the tumor are easily recognizable.

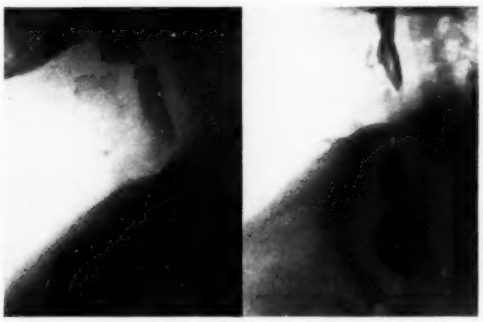

Fig. 12. A very long pharyngeal tumor. The picture at the left shows the upper pole with a part of the stricture. The picture on the right was taken at a little later phase of the swallowing act, and shows the lower pole with the stenosis above it. The upper pole is at the level of the epiglottis; the lower is below the upper sternal margin.

the upper pole which is still situated at the level of the epiglottis, i.e., in the mesopharynx, and a part of the stricture. The picture on the right, taken at a later phase of the act of swallowing, demonstrates the lower pole of the tumor which is located in the œsophagus, two fingerbreadths below the upper thoracic aperture, and the stricture just above it. The tumor involves the anterior wall of the pharynx and the cervical section of the œsophagus. There are no changes in the trachea or the spinal column.

It is evident that X-ray examination can afford us information only on the extent of the *protrusion* of a tumor, which is seen in the picture as a defect in the contrast shadow. In other words, the tumor is recognizable only in so far as it is prominent. A simple infiltration of the wall cannot be recognized by means of X-ray examination. Nevertheless X-ray examination is of great value in locating the lower pole of a tumor of the pharynx and thus in helping us to decide on the operative treatment.

SUMMARY

In incomplete stenoses of the œsophagus, X-ray examination with the patient in horizontal position furnishes us important information on the length and form of the stenosis. Since, in horizontal position, the passage of the paste through the œsophagus is retarded, the latter is well filled within and below the point of stricture as well as above the stenosis, and this enables us to orient ourselves on the length and shape of the stricture, contrary to the possibilities afforded by examination of the patient in the upright position. This knowledge is especially important in the case of cicatricial strictures of the œsophagus because it affords great help in endoscopic treatment. The diagnosis of diverticula, particularly of traction diverticula, is also enhanced by this method.

This method of X-ray examination is also valuable for the diagnosis of tumors of the mesopharynx and hypopharynx, as the diagnosis of these growths in upright position is quite uncertain. As a result of the horizontal position, the passage of the paste swallowed is retarded, and there is time for a more complete filling of the pharyngeal cavity. The defect within this picture indicates the size of the tumor, and we can see the entire extent of the neoplasm in so far as it protrudes into the pharyngeal lumen. Since, in general, laryngological examination cannot reveal the length of the neoplasm, the knowledge gained by such a method of X-ray examination doubtless increases our ability to determine with greater nicety whether further treatment (operative or conservative) is indicated, and broadens the horizon of our diagnostic ability.

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ENLARGEMENT OF THE PARTURIENT CANAL BY BONE GRAFT[1]

By FRED H. ALBEE, M.D., Sc.D., F.A.C.S., New York

REASONING *a priori* from extensive experience in the modification of the contours and dimensions of the skeleton, enlargement of the pelvis by bone graft to increase the parturient diameters presents no more difficult a mechanical problem than many which are daily solved in the operating room of the bone-plastic surgeon. The feasibility of this procedure has long appealed to the author; but as he is not an obstetrician, opportunity to work up any considerable series of cases has been lacking.

Two cases are reported in which the author has enlarged the pelvic diameters by placing an inlay graft between the symphyses pubis, after forcing them apart 1½ inches. In both cases the desired result was obtained: the ability to bear children by normal delivery.

REPORT OF CASES

Case 1. A woman, aged 30 years, unable to bear children because of a contracted pelvis, was referred to me in October, 1916, to consider the possibility of enlarging the pelvis by bone graft.

A horizontal incision was made over the symphysis pubis, the pubic ligaments sectioned, and the innominate bones separated at the symphysis. A graft was then taken from the tibia, by the same technique used in other bone-grafting operations, and inserted in the hiatus between the innominate bones of the pelvis. The graft was then sutured *in situ* with kangaroo tendons, and the wound closed in the usual way.

The following year the young woman became pregnant and gave birth to a normal, healthy infant.

Case 2. E. J., a woman, aged 34 years, had a pelvis so small that normal delivery was impossible. She had been pregnant once, and the child was delivered at full term by craniotomy. At this time she was told that she could not have children except by cæsarean section because of her contracted pelvis.

Within a year she again became pregnant, and 6 months later (March 1, 1923) was referred to me by the late Dr. Samuel Druskin, a well known obstetrician, to see whether or not it was possible to increase the pelvic outlet. March 29, 1923, she was admitted to the Postgraduate Hospital.

The fact of pregnancy naturally increased the difficulties of operation, but in spite of this the recovery was uneventful, and the patient later delivered a normal child. She has since borne two other healthy children by normal delivery. The technique used in this case was the same as that in Case 1 (Figures 1 to 6).

I am greatly indebted to the late Dr. Druskin not only for referring the case, but for his encouragement in going forward with the operation during pregnancy.

The author believes that the graft method possesses distinct advantages over symphysiotomy and pubiotomy.

In both cases reported, considerable pressure had to be exerted by a specially designed spreader in order to increase forcibly the size of the pelvis, and after gaining the increase, to hold the bones apart while the graft was being inserted. Judging from the author's experience in these two operations, the force required to separate the bones any appreciable amount is very great, and after symphysiotomy this force must come wholly from the wedging effect of the head as it is forced downward by the muscles above.

Another advantage of the inlay graft method as compared to symphysiotomy and pubiotomy is that the latter may have to be repeated at each pregnancy, in that the hiatus temporarily secured

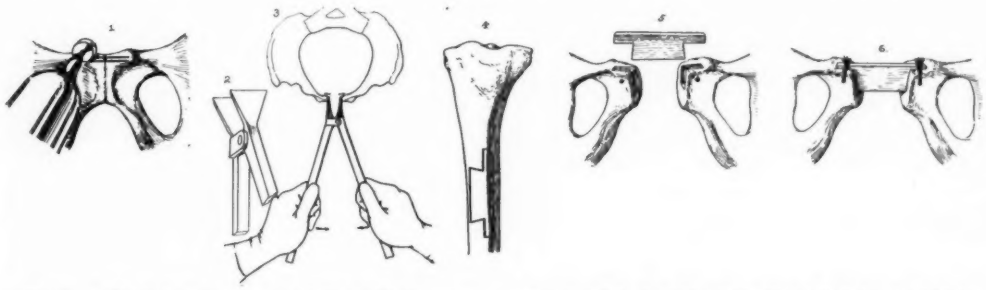

Fig. 1. Author's twin saw grooving the pubic bones on either side of the symphyses.

Figs. 2 and 3. Spreader used for spreading pelvis after the severance of the symphyses.

Fig. 4. Shape of graft obtained from tibia.

Fig. 5. Bed of graft. Note the drill holes for the insertion of heavy kangaroo tendon; also the graft ready to be inlaid.

Fig. 6. Tibial graft inserted, with kangaroo-tendon sutures in place.

[1]Submitted for publication, November 24, 1927.

during labor immediately disappears. The author has repeatedly confirmed this by X-ray study of cases immediately and remotely following operation. On the other hand, the enlargement of the pelvic ring by bone graft is a permanent enlargement. The only alternative is cæsarean section.

The inlay graft is apparently the only method which effects permanent enlargement of pelves too small for normal delivery. It is particularly applicable to the juxtominor type.

TECHNIQUE

The technique is shown in Figures 1 to 6. The ends of the graft are mortised into the symphyses by the author's inlay technique used in other bone-grafting operations. In the enlarging of the pelvis, the strength of the graft is exceedingly important. Great care must be taken that it is sufficiently strong to withstand the pressure of the bones as they attempt to get back to their former position.

STENOSING FIBROUS TENDOVAGINITIS OVER RADIAL STYLOID (DE QUERVAIN[1])

By CHESTER C. SCHNEIDER, M.D., Milwaukee, Wisconsin

FOR several years my attention has been directed to an interesting condition at the wrist concerning which I could find no enlightenment in American literature. The relative frequency of the condition and the striking similarity of all cases in their symptoms and physical findings made it seem evident that this was a definite clinical entity worthy of careful study and analysis.

The patients with this condition invariably complain of severe pain at the wrist, more or less definitely localized in the region of the radial styloid, and radiating into the hand and up the forearm. They give a history of a gradually developing pain at the site of the radial styloid usually without any precipitating trauma. This pain becomes so severe in the course of weeks or months that complete disability of the wrist ensues. Occasionally the pain assumes a neuralgic character and is very severe at night. Abduction and extension of the thumb are accompanied by severe pain, and pressure over the radial styloid is exquisitely tender. Practically all of the patients that have come under my care have been seen previously by one or more physicians who have variously diagnosed the condition as rheumatism, neuralgia, and neuritis, usually prescribing heat, salves, electricity, and rest. Despite these measures, the symptoms have become increasingly severe, finally incapacitating the wrist for further use.

Physical examination reveals definite swelling in the region of the radial styloid (Fig. 1.) extremely tender to pressure. Abduction and extension of the thumb are very painful, in many instances the patients being unable to perform this motion at all. Even gripping and ulnar and dorsiflexion produce pain over the involved region. Frequently a gristle-like thickening can be felt under the skin while at times the periosteum of the underlying radius seems thickened. There is never any local heat, redness, infiltration, or *crepitation* in the tendon sheath on motion.

It is interesting to note that all the pathology occurs at the point where the abductor pollicis longus and extensor pollicis brevis tendons pass under the superficial dorsal carpal ligament through the osteofibrous canal immediately overlying the radius. Because of the fact that these muscles arise on the dorsal surface of the forearm as extensors and curve around the distal end of the radius to become abductors of the thumb (Fig. 2.), they are exposed to increased friction at this site. Likewise, in consequence of their superficial position between the bone and skin they become exposed to more frequent trauma than other tendons.

This condition was first described in 1895 by de Quervain, of Basle, Switzerland, who reported 5 cases in the *Correspondenz-Blatt fuer schweizer Aerzte*. He saw his first case under Professor Kocher at the Bern Klinik in 1893. Subsequent to de Quervain's first paper, a considerable number of German and French clinicians reported additional cases, investigating the condition quite exhaustively. The most comprehensive study was made by Alfons Eschle, of Basle, in 1924 who tabulated the 110 cases previously reported in the literature and added 19 more which had come under his own observation.

[1] Read before the Milwaukee Academy of Medicine, October 11, 1927.

Fig. 1. Photograph of normal and involved wrists showing swelling over radial styloid in the latter.

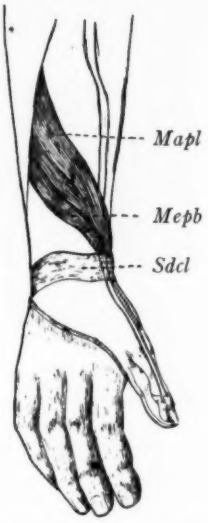

Fig. 2. Sketch showing origin and insertion of abductor pollicis longus and extensor pollicis brevis. Note how tendons curve around radial styloid in common tendon sheath under superficial dorsal carpal ligament. *Mapl* abductor pollicis longus muscle; *Mepb*, extensor pollicis brevis muscle; *Sdcl*, superficial dorsal carpal ligament.

Although I have personally seen and treated between 25 and 30 cases in clinical and private practice, I have been able to collect the records of only 15 of these. This makes a grand total of 144 cases. In 133 of these where the sex was reported, 119 were women and 14 men. Out of 89 where the side was noted, 47 were right, 44 left, and 4 bilateral. These patients ranged in age from 15 to 75 years, the majority, however, being between 35 and 55. Housewives and maids constituted the largest vocational group, there being 81 with this classification. Further, there were 7 farm women, 4 factory workers, 3 nurses, 2 pianists, 1 milliner, 1 plasterer, 1 hairdresser, 1 laborer, and 1 architect.

Although the exact *etiology* of this condition is not clear, there seems to be more or less agreement among the various authors that *excessively monotonous* use of the involved tendons is the most commonly evident cause. The incidence of the disease practically exclusively among workers who use their thumbs a great deal seems to substantiate this view. In some instances a single direct blow to the exposed tendon sheath has been the sole etiological factor, while in other cases injury followed by excessive use of the thumb has been responsible. A few authors have noted a rheumatic predisposition in some of their cases, but this is by no means constant. Lues and tuberculosis have been definitely ruled out as bearing no relationship to this condition. The primary cause, then, as far as can be determined, is a mechanical rubbing of the abductor pollicis longus and extensor pollicis brevis tendons over the rounded distal end of the radius. This becomes quantitatively increased by increased force or frequency and produces an œdema of the tendon sheath and results in a vicious circle, increasing the friction and causing the lumen of the tendon sheath to become narrowed. Trauma qualitatively influences the irritation by increasing the œdema or changing the underlying bone.

According to all the authors who have previously written upon this subject, X-ray examination has consistently failed to reveal any change in the underlying bone. My own cases have corroborated this observation with one or two exceptions. Figure 3, a roentgenogram of the normal and involved wrists, reveals some deposition of lime salts over the radius at the site of the fibrocartilaginous disc of the diseased tendon sheath. This suggests a probable extension to the periosteum of the bone in the nature of a proliferative reaction such as one might expect to see following trauma or inflammation.

Figure 4 shows a similar change, but to a lesser extent in another case. These bone changes might be ascribed to the extension of irritation to the periosteum or may be only structural variations in normal bones.

No relationship has been established between this disease and syphilis. All the blood Wassermann tests in my own cases have been negative as is true of all the cases previously reported in the literature. Likewise, no evidence of tuberculosis has been noted in these cases. Although tuber-

Fig. 3 (left). X-ray of normal and involved wrists showing deposition of lime salts over radial styloid.

Fig. 4. X-ray of another case of de Quervain's disease showing slight deposition of lime salts over radial styloid.

culous tenosynovitis is a well known disease, tendovaginitis of the de Quervain type has never been shown to be tuberculous and does not in any way follow the course of the tuberculous cases.

The pathology of this condition has been studied very carefully by Kocher, de Quervain, Keppler, Vischer, Hauck, Nussbaum, Flörcken, and Eschle. It was Kocher who first named the condition "stenosing, fibrous tendovaginitis" because of the fibrous thickening which he noted in the middle layers of the tendon sheath. He looked upon the condition as a *passive* function hypertrophy and placed it in the same class with snapping finger (*doigt à ressort*). According to Nussbaum, the normal tendon sheath is composed of 5 layers. The inner layer is the parietal part of the same tissue which covers the tendons (visceral layer) and is composed of 2 strata of flat or cubical cells with spindle-shaped or round nuclei. This layer frequently has finger-like processes and contains fine lymph vessels without muscle walls. The second layer is composed of loose connective tissue containing nerves and blood vessels. Then follow 2 thin, firm layers of connective tissue, the inner layer with fibers running parallel to, the outer across, the blood supply, the latter being the wider of the two. The fifth and last layer is composed of loose connective tissue with nerves and blood vessels. Whereas grossly the common tendon sheath of the abductor pollicis longus and extensor pollicis brevis tendons is normally a thin, colorless, transparent membrane, in this disease, it is thickened, non-transparent, and cuts like gristle. The canal is narrowed so that in some cases it is difficult to pass a thin probe through it. When cut, the sheath looks milk white, the inner surface being yellow to red in color, here and there being velvet-like. While at times the sheath itself may be thickened to 4 times its normal depth, the tendons themselves show no changes. Occasionally the 2

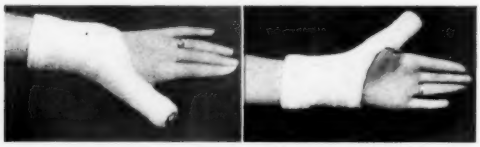

Fig. 5. (left). Photograph of immobilization cast employed by author.

Fig. 6. Same cast as in Figure 5, side view.

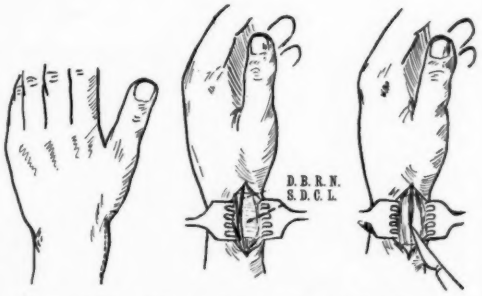

Fig. 7 (left). Sketch of wrist, dotted line indicating site of incision in skin.

Fig. 8 (center). Second stage of operation showing dorsal branch of radial nerve which is to be avoided. *D. B. R. N.*, dorsal branch of radial nerve; *S. D. C. L.*, superficial dorsal corpal ligament.

Fig. 9 (right). Third step of operation showing splitting of tendon sheath of extensor-abductor tendons to thumb.

TABLE I.—SYNOPSIS OF FIFTEEN CASES OF DE QUERVAIN'S DISEASE PERSONALLY REVIEWED

No.	Name	Age	Occupation	Side	Duration	Cause	Treatment		Result
1	V. F.	50	Milliner	Left	1 month	Arthritis	Cast 2½ weeks	Further Rx refused.	Unimproved
2	F. B.	30	Laborer	Right	5 months	Excessive use	Cast 6 weeks Operation		Unimproved Cured
3	C. B.	48	Architect	Right	2 weeks	Excessive use	Splint 2 weeks	Further Rx refused	Unimproved
4	M. R.	15	Student	Left	1 year	Baseball strain	Cast 4 weeks		Cured
5	E. B.	21	Dressmaker	Right	1 month	Sewing strain	Cast 6 weeks		Cured
6	C. G.	16	Student		1 month	Using crutch	None		
7	K. F.	33	Housewife	Right	6 months	Excessive use	Cast 6 weeks		Cured
8	S. T.	50	Housewife	Left			Cast 6 weeks		Cured
9	V. J.	25	Housewife	Left	3 weeks		Cast 6 weeks		Cured
10	C. W.	47	Housewife	Right		Arthritis	Cast 5 weeks		Cured
11	F. D.	40	Nurse	Right	2 weeks	Excessive use	Splint 6 weeks		Cured
12	C. P.	37	Nurse	Right	2 months	Excessive use	Cast 6 weeks		Cured
13	A. B.	40	Housewife	Right	2 months	Excessive use	Cast 6 weeks Operation		Unimproved Cured
14	C. F.	45	Nurse	Left	5 months	Lifting invalid mother	Cast 6 weeks	Further Rx refused	Unimproved
15	J. M.	53	Housewife	Left	3 months	Fall on wrist	Untreated		

tendons may be adherent to each other with a thin, fibrous band. Three of Keppler's cases showed firm adhesions between the tendons and the sheath. All the authors seem to agree that the tendon sheath is too narrow for the tendons.

Histologically most of the cases fall into one of two groups, according to Nussbaum. Group 1 in which the majority of them lie, shows a connective tissue proliferation in the sheath 3-fold to 4-fold. There is no fixed direction of the fibers and the endothelium and its substratum are frequently absent. In the second group, the blood vessels and nerves, loosely held in the outer layers of the sheath, are surrounded by round-celled infiltration. Many sections in this region show small, irregular, necrotic areas. On the inner side of the sheath the blood vessels are increased. The occlusion of the lumen of the sheath is often directly produced by necrotic masses. This histological evidence would point to the fact that we are here dealing with a reaction to trauma or excess use which is not inflammatory. Some factor is responsible for the stimulation of the fibrous tissue growth in the connective tissue layers of the tendon sheath.

In the treatment of this condition, a wide field of therapeutic aids has been tested, practically all of them to no avail except prolonged immobilization and surgery. Heat, electricity, splints, rest, massage, internal medicaments, etc., have been of no avail. Practically all of the European writers, who have described this condition, are agreed that conservative measures do not yield encouraging results. In my own practice I have employed a plaster-of-Paris cast to the thumb and wrist with the thumb in full extension and abduction (Figs. 5 and 6). Such casts when allowed to remain on from 4 to 6 weeks produced a complete remission of symptoms in 7 out of 10 cases. Immobilization must be absolute and sufficiently prolonged to permit complete resolution of the pathological changes which have taken place in the sheath. In those cases where the fibrous stenosis has become too advanced to respond to immobilization alone, surgery has provided a simple and effective solution to the disability. The operation proposed by de Quervain and successfully used by a number of European surgeons is as follows: Under a local anæsthetic, a linear incision about 1½ inches long is made over

the distal end of the radius through the skin and subcutaneous tissue. Overlying the superficial dorsal carpal ligament is a small branch of the radial nerve which should be retracted. A cut is made through the carpal ligament immediately over the tendon sheath and then the thickened sheath is split for a distance of about 1 inch. The skin is closed and the wound dressed without a cast. Figures 7, 8 and 9 show the steps of the operation. Active motion can be undertaken within a few days and full restoration of function may be anticipated within 2 or 3 weeks. Of 66 cases collected by Eschle which were treated surgically in this manner, 65 were cured and 1 improved. I have resorted to surgical division of the common tendon sheath in 2 cases, both of which were *cured*.

Table I gives a summary of the 15 cases from my own records and indicates the treatment rendered and results obtained, along with other pertinent data. It will be seen from a cursory examination of this that the correct employment of the plaster cast has afforded cures in 70 per cent of the cases thus treated and that this treatment is to be recommended in the cases seen fairly early.

To summarize briefly, we are here dealing with a clinical entity, not previously described in American literature or textbooks, known as de Quervain's disease. This is a passive function hypertrophy of the fibrous layers of the common sheath of the abductor pollicis longus and extensor pollicis brevis tendons causing a stenosis of the sheath over the radial styloid and impairment of function of the thumb. The pain over the radial styloid may be so severe as to produce complete disability at the wrist. The condition is a chronic one and responds only to complete immobilization of the thumb and wrist in a cast from 4 to 6 weeks, or in late cases, surgical splitting of the stenosed tendon sheath.

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SUBPERITONEAL CHOLECYSTECTOMY[1]

By LESTER R. WHITAKER, M.D., Rochester, New York
National Research Council Fellow under Professor John J. Morton

ANY abdominal operation should attain its object with as little trauma of the viscera as possible to the end that shock may be kept at a minimum, postoperative discomfort reduced, convalescence shortened, and adhesions prevented. Exposure of the cystic duct in the usual operation for cholecystectomy necessitates a great deal of gauze packing, with pressure and traction on the viscera, which renders the operation more dangerous and more likely to be followed by complications than is perhaps necessary.

To obviate these disadvantages the following method has been developed, thus far applied only experimentally to dogs, though for anatomical reasons it should not be more difficult in selected patients.

The principle of the operation is removal of the gall bladder through a small abdominal incision by subperitoneal dissection from above downward to the cystic duct.[2] This depends upon the anatomical fact that the peritoneal covering is very loosely attached to the gall bladder. I have frequently noted the empty, contracted gall bladder of an animal to be rolled up beneath its serosa, which is attached along the sides of the gall-bladder fossa of the liver. This relation has been observed also in patients at necropsy, following manual expression of the vesicular content. The serosa thus acts as a covering or cloak under which the viscus may contract or move about. Its laxity is of extreme importance in the operation to be described, not only insuring ease of separation but allowing sufficient retraction of the membrane away from the gall bladder to obtain exposure, even as far down as the cystic duct.

TECHNIQUE

An incision is made as nearly over the gall bladder as possible, only large enough to allow delivery of the fundus. Small retractors on opposite sides lift the abdominal wall upward and outward; and if the gall bladder is not immediately visible, an exploratory lamp is inserted with a curved blade attached to one side which acts as a visceral retractor. Upon sight, the fundus of the gall bladder is grasped with a triangular-tipped Collins clamp and pulled into the wound (Fig. 1). The two retractors are then replaced by a small self-retaining retractor. A circular incision is made with a very sharp knife through the serosa near the tip of the vesicle (Fig. 2). By means of fine forceps and a rather sharp round-tipped suction dissector[3] the serosa is freed from the gall bladder for 1 or 2 centimeters, thus forming a peritoneal cuff (Fig. 3). Silk traction sutures applied radially to the edge of the cuff are secured to the drapings, or held by assistants, thus opening the cuff. Traction on the gall bladder is maintained. By the aid of a headlight and a special wire retractor, and using either the suction dissector or gauze tips, the dissection is carried down toward the cystic duct (Fig. 4). If distended, the gall bladder can be opened and partially emptied of its contents in order to facilitate dissection.

[2] In 1899 Doyen removed the gall bladder and drained subperitoneally but through a regular incision, with large retractors and gauze packing for exposure. Doyen: Surgical Therapeutics and Operative Technique. 1920 ed. iii, 121. Since then numerous modifications have been made:
Yates, J. L. Indications for cholecystectomy and a method for performing it. Surg., Gynec. & Obst., 1920, xxx, 514.
Andrews, E. Wyllys, and E. Extraperitoneal drainage of a cholecystectomy wound. Surg. Clin of N. A., 1921, 1197.
Gibson, C. L. Aids to cholecystectomy. Ann. Surg., 1926, cxxxiii, 618.
Foster, G. S. The technique of cholecystectomy. Am. J. Surg., 1927, iii, 144.

[3] This suction dissector was invented by Dr. Clifford B. Walker and has been used in the Surgical Clinic of the Peter Bent Brigham Hospital for the removal of tonsils.

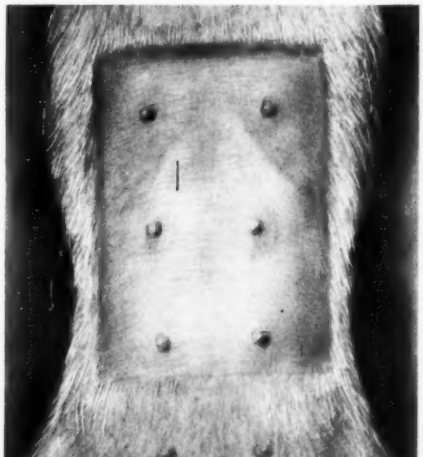
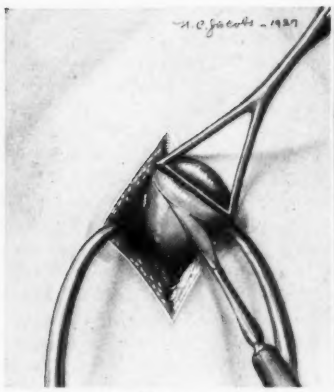


Fig. 1. Showing position and relative size of incision for subperitoneal cholecystectomy in a dog.

[1] Department of Surgery, The University of Rochester School of Medicine and Dentistry, Rochester, New York. A preliminary report was published in the Boston Medical and Surgical Journal, 1927, cxcvii, 657.



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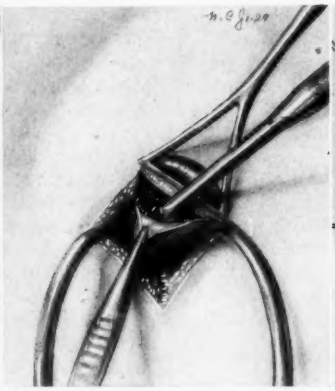

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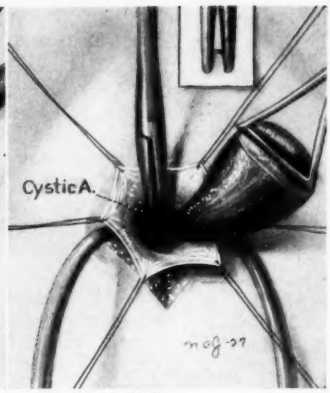

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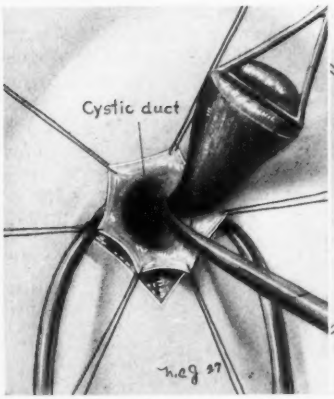

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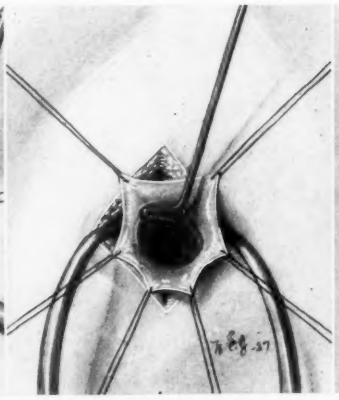

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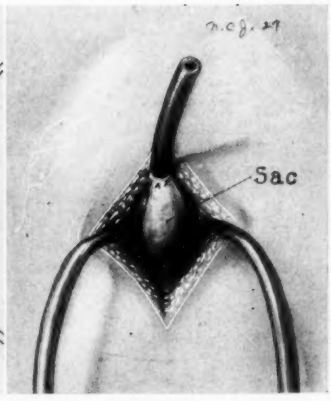

Fig. 7.

Fig. 2. Gall bladder held up by a Collins clamp; self-retaining retractor in place; a circular incision being made through the peritoneum. The drapings have been omitted so that the field may be clearly seen.

Fig. 3. Beginning separation of the peritoneum with forceps and suction-dissector.

Fig. 4. Radial traction sutures in peritoneal sleeve. Silver clip being placed on cystic artery, which, for the purposes of illustration, the artist has drawn more clearly than it appears.

Fig. 5. Cystic duct dissected free, clamped, and tied.

Fig. 6. Showing sac from which gall bladder was removed being held open by a special wire retractor. The stump of the cystic duct has retracted into the hole at the bottom.

Fig. 7. Sac collapsed about rubber drainage tube.

Ordinarily, however, the process is easier with a moderately full vesicle.

When resistant strands of fibrous tissue interfere with the stripping process they are cut with scissors close to the gall bladder in order to avoid "button-holing" the peritoneal sleeve or damaging the liver. If the strands be large, a Cushing silver clip should previously be applied, for some of them will contain vessels running from the gall bladder to the peritoneum. Surprisingly little bleeding is encountered, however, since most of the vessels remain on the gall bladder. Should any be torn off, the bleeding points on the vesicle can be stopped by a touch with the electrocoagulator or by a silver clip. The suction dissector keeps the field clear of blood oozing from the small vessels. Near the region of the cystic duct, all large strands of fibrous tissue should be doubly silver-clipped before division, since one of them will contain the cystic artery (Fig. 4). If the artery by chance breaks loose, there is little danger since all the bleeding is within the peritoneal

Fig. 8. Sutured wound with drainage tube.

sleeve. The blood can be removed with a sucker of adequate size and time taken to secure the vessel. If this is not possible, a gauze packing kept dry with suction can be applied until coagulation occurs. In some of my experimental operations in which the cystic artery broke loose and was not secured, not over 50 cubic centimeters of blood was lost.

After the cystic artery is cut, the cystic duct is easily dissected out under traction. If the duct breaks off during this process, no damage ensues, since any leakage of bile will be carried off through the peritoneal sleeve. When the duct is freed, it is clamped as low as possible, tied, and the vesicle removed (Fig. 5). Upon release, the stump retracts into the recess under the peritoneum from which the duct had been withdrawn (Fig. 6).

Should there be a question of common bile-duct obstruction in patients, however (though this should have been ruled out by careful preoperative examination), iodized oil can be injected into the stump of the cystic duct, left rather long, and roentgenograms of the ducts made (Fig. 9). If it should be expedient to test the patency of the ducts before this stage is reached, the gall bladder can be opened, emptied of its contents, and filled with iodized oil; then by gentle pressure, the oil can be forced down the ducts. If unforeseen obstruction is noted, it can be dealt with in the usual manner if the incision is enlarged.

Finally, after subperitoneal removal of the gall bladder, a small rubber tube with side holes is placed in the peritoneal sleeve and brought to the outside, the sleeve being collapsed about the tube and lightly tied at the point of contact with the abdominal wall (Fig. 7). Thus the tube is completely peritonealized and any leakage of bile or blood into the peritoneal cavity precluded.

Fig. 9. Roentgenogram of the biliary passages of a dog. Iodized oil was placed in the gall bladder and gentle pressure exerted. Cut shows a small amount of oil in the gall bladder (above), common duct full, and a large amount in the duodenum (below).

The operation has been performed upon 16 dogs. Fourteen made perfect recoveries, apparently showing very few effects of any kind from the procedure. Two died after the operation, one of peritonitis and bile leakage, in which the cystic duct had been clamped and electrically coagulated, no drainage of the peritoneal sac being employed; the other, also not drained, died of leakage of blood and bile into the peritoneal cavity through an accidental opening in the peritoneal sac. These two poor results emphasize the necessity for stopping hæmorrhage before closing the wound and for subperitoneal drainage for 1 to 2 days. The 14 other dogs at necropsy showed only a few omental adhesions at the operative site.

THE QUESTION OF CLINICAL APPLICATION

The first requisite for such a procedure as has been described would be a definite diagnosis. It must be determined with reasonable certainty

that the gall bladder and not the stomach, duodenum, or pancreas is the organ diseased. This implies extremely careful study of the case and the application of all necessary tests including roentgenological studies of the gastro-intestinal tract and frequent determinations of the icterus index over a period of one or more weeks.

In the diagnosis of gall-bladder disease, the history is of great significance, and the ruling-out of disease of other organs by appropriate tests is essential, but cholecystography is the method *par excellence*. If a shadow of the gall bladder be obtained, the condition of the viscus can be accurately predicted in almost every instance. A shadow definitely mottled in successive films indicates stones, while contraction after feeding indicates an active musculature without marked sclerosis. This combination frequently occurs. Furthermore, the very production of a shadow implies that the vesicle is active enough to empty and allow refilling, indicating a nonsclerotic wall and a serosa which will readily strip away. Such a gall bladder presents the ideal case for subperitoneal cholecystectomy. The method would probably have been applicable to all the cases reported in my article on the diagnosis of gall-bladder disease.[1]

If no shadow of the gall bladder be obtained by the intravenous method of cholecystography, the gall bladder is almost surely diseased, perhaps severely enough to preclude subperitoneal cholecystectomy; but in many cases the failure of shadow formation is due to partial blockage of the cystic duct with stones, or a gall bladder so full of stones that an insufficient quantity of the radio-opaque material can enter; yet in either of these cases the wall may not be sclerotic enough to prevent stripping of the serosa.

To determine whether or not this be true, a small incision can be made as nearly over the fundus of the gall bladder as may be after very careful percussion and palpation with the patient under ether and the condition investigated by an exploratory lamp. If the gall bladder be found fibrotic or bound down by a mass of adhesions, of course the case is unfavorable for subperitoneal cholecystectomy and the regular operation should be done. The presence of a few adhesions need not necessarily contra-indicate a subperitoneal cholecystectomy. The gall bladder may be pulled up, if possible, and the adhesions ignored, or they may be previously dissected away. I have observed that in many excised gall bladders which had had adhesions, the serosa would still strip readily. Subperitoneal cholecystectomy cannot be done if the gall bladder is too friable to allow moderate traction.

[1] Whitaker, L. R. The diagnosis of gall-bladder disease. The application of recent findings in physiology. J. Am. M. Ass., 1927, cxxxviii, 1791.

For assistance in this work the writer is indebted to his associates, Drs. Lynn C. Fort, David W. Pratt, William C. Emerson, and Oscar W. Simpson; and to Mr. Natt C. Jacobs, artist.

A PRACTICAL CONSIDERATION OF THE DAMAGED PELVIC FLOOR WITH A TECHNIQUE FOR ITS SECONDARY RECONSTRUCTION[1]

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Attending Surgeon, Woman's Hospital

THE operations which are commonly employed in the secondary reconstruction of the lacerated or relaxed pelvic floor are of two distinct types. The less radical type of operation which consists of a superficial denudation of vaginal mucous membrane, an inversion of the denuded portion of the rectovaginal septum, and a reconstruction of the perineum is still used by a considerable number of both gynecologists and general surgeons. The majority of operators, however, have adopted a more radical type of procedure in which some form of flap-splitting of the rectovaginal septum is followed by a direct union of the anterior portions of the levatores ani muscles between the vagina and rectum and by a reconstruction of the septum and perineum.

During the past decade many operations of both types have been performed according to the accepted technique by members of the staff of the Woman's Hospital, and in approximately 75 per cent of the cases, the end-results have been carefully observed for periods of time ranging from 1 to 3 years. The follow-up findings have thrown much light upon the relative merits of the two generally accepted methods of reconstructing the damaged pelvic floor and have clearly revealed certain objectionable features in the end-results of both types of operation.

The objections to all operations in which denudations and inversions of redundant tissue are employed are (1) the impossibility of a restoration of the levator ani muscle to normal anatomical position and function by such procedures, and (2) the unjustifiable incidence of recurrent rectocele which follows their application.

In the restoration of the retracted levator ani to its normal position and function, it is essential to expose its fascial covering and the superior layer of the urogenital trigone to which its fascia is attached. To do so necessitates flap-splitting of the rectovaginal septum, and a type of flap-splitting in which the flap includes all layers of the septum from the vaginal mucosa to the rectal musculature. An examination of the accompanying anatomical charts convinces one of the impossibility of such an exposure by any form of mucous membrane denudation.

The high incidence of recurrent rectocele which follows the type of operation under consideration is satisfactorily explained by the fact that in this type of procedure the fundamental principles which underlie a successful surgical correction of a hernia—and a rectocele is a hernia—have not been observed. In the surgical correction of any hernia, it is of fundamental importance to incise all layers of the hernial sac, which in a rectocele is the herniated portion of the rectovaginal septum, and to remove from the sac its content, the abnormally situated and adherent rectum in the case of the rectocele. The remainder of the correction consists of the removal of the hernial sac and the closure of the hernial opening through the use of available muscles and fascia. It is obvious that such principles are not observed in the superficial type of operation.

The more radical operations in which flap-splitting and direct levator myorrhaphy are employed, though productive of generally good results in the cure of rectocele, fail in most instances to restore the levator ani to normal function. As levator myorrhaphy is usually performed, varying amounts of the fascial-covered and immobilized anterior fibers of the muscle of one side are united between the vagina and rectum with a similar bundle of fibers from the muscle of the opposite side. Sutures which have been placed about the muscle bundles above the perineum are tied under a tension sufficient to hold them together until union occurs. Despite the care with which such sutures are tightened, it is extremely difficult to avoid a pressure atrophy of the included muscle fibers with the result that a more or less crescentic, rigid, and functionless band of scar tissue forms at the site of the junction of the muscle bundles. It is a well recognized fact that such bands of scar tissue cause dyspareunia and dystocia, and are responsible for an impairment or complete loss of function in the most important fibers of the levator muscles.

In an effort to avoid the objectionable sequelæ of the two conventional methods of reconstructing the injured pelvic floor the operation herein described was devised. It has been under trial by several members of the staff of the Woman's Hospital for the past 4 years, and has gained a confidence which seems to warrant a preliminary report at this time. It has been based upon a recognition of the following facts concerning the normal anat-

[1]From the Clinic of the Woman's Hospital.

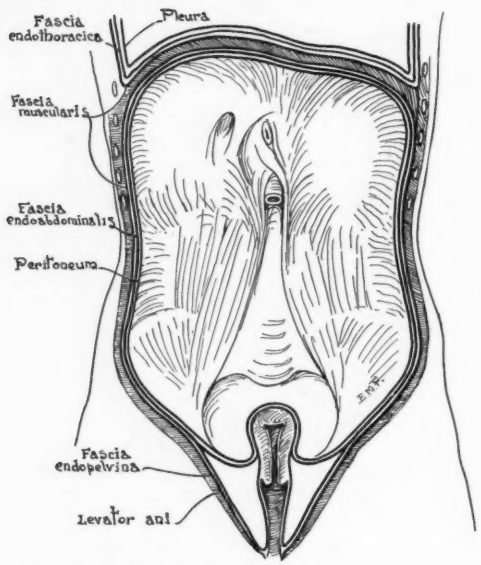

Fig. 1.[1] Illustrating the relationship between peritoneum, the fascia endo-abdominalis, the fascia endopelvina, and the fascia muscularis.

omy and function of the pelvic floor and the changes which occur as a result of injury at the time of parturition.

NORMAL ANATOMY OF THE PELVIC FLOOR

The pelvic floor or pelvic diaphragm is formed by the levatores ani muscles, the coccygeus muscles, and the fascia which surrounds them. The diaphragm thus formed almost closes the pelvic outlet and separates the pelvic cavity from the perineum which completes the closure.

The levator ani muscle, the larger and more important of the two muscles which form the muscular portion of the pelvic diaphragm, arises from the posterior surface of the os pubis, the white line (arcus tendineus musculi levatoris ani) and the spine of the ischium. From this broad origin, the fibers run downward, backward, and inward to become attached to the sides and tip of the coccyx, to the fibrous raphe (ligamentum anococcygeum), which extends from the tip of the coccyx to the anus, and to the sides of the rectum. A few fibers, the fibers of Luscka, from the most anterior portion of the muscle, pass between the vagina and rectum to be inserted into the perineal body.

Though the levator ani is usually regarded as one muscle it is composed of three separate, flat

[1] Figures 1 to 5 are redrawn from Halban.

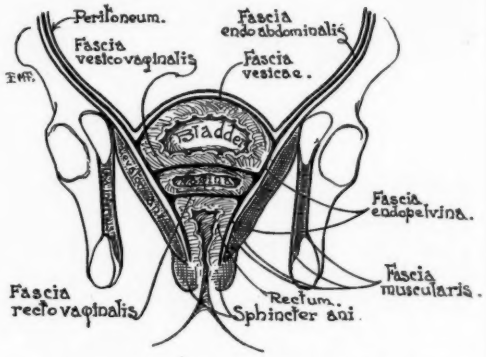

Fig. 2. Illustrating the distribution of the fascia endopelvina about the bladder, the vagina, and the rectum.

segments. Piersol (7), in commenting upon this phase of the subject, says: "There is usually to be found a dividing line across the muscle on a level with the junction of the superior ramus of the pubis with the ilium and separating those fibers which are inserted into the coccyx and the posterior portion of the fibrous raphe from those which pass to the anterior part of the raphe and the rectum. Each of the portions so separated is supplied by a separate nerve and this, combined with the results of comparative anatomy, shows that the posterior portion of the levator is really a muscle quite distinct from the anterior portion. It has been termed the musculus iliococcygeus. Furthermore, it seems probable that the anterior portion is composed of two morphologically distinct muscles, one of which arises from the pubis and anterior part of the white line and is inserted into the median fibrous raphe, whence it is termed the pubococcygeus; while the other, situated beneath, i.e., superficial to the pubococcygeus, consists of those fibers which arise from the pubis and are inserted into the rectum, and is termed the musculus puborectalis."

There has been considerable controversy concerning the presence or absence of fibers of the levator ani in the perineal body between the rectum and the vagina. Dissections by Martin (6) and others have proved conclusively that there are fibers of the puborectalis, the fibers of Luscka, which pass between the rectum and vagina to insert into the perineal body.

Of the three portions of the levator ani, the two more anterior portions, the pubococcygeus and the puborectalis, are, from the viewpoint of the gynecologist and obstetrician, the most important segments both structurally and functionally. In commenting upon the pubic segments, Sturmdorf

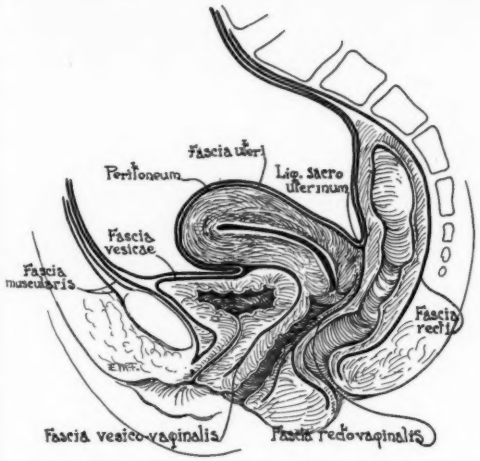

Fig. 3. Illustrating in sagittal section the distribution of the fascia endopelvina about the bladder, the vagina, and the rectum.

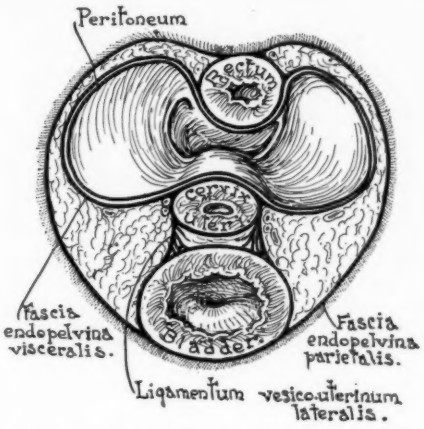

Fig. 4. Illustrating in a cross section made at a level of the cervix, the distribution of the fascia endopelvina.

(9) has said: "Their lines of origin extend for 1½ inches on either side of the posterior surface of the pubic symphysis, thus equaling in width the average sternomastoid; they are twice as thick as the diaphragm, weigh one-fourth as much as the external oblique, altogether presenting a muscular support exceeding that guarding the inguinal ring.

"These pubic segments course almost horizontally backward and inward along the lateral vaginal wall. They converge rapidly toward those of the opposite side to become inserted into the perineal body, the rectal walls and anococcygeal tendon, encircling the vagina and rectum in distinct loops.

"Their median borders, which are plainly palpable through the lateral vaginal walls, a half inch or less behind the plane of the hymen, form a V-shaped interspace which embraces the introitus under the pubic arch, and is termed the levator cleft (hiatus genitalis). In this cleft are the urethra, vagina, and rectum."

The coccygeus muscle, the flat, fan-shaped muscle which completes the muscular pelvic diaphragm posteriorly, arises from the spine of the ischium and is inserted into the sides of the sacrum and coccyx. It is of no practical importance to the gynecologist.

The pelvic fascia, the fascia of the pelvic diaphragm, is a continuation of the iliac fascia. It descends over the anterior surface of the pyriformis, the upper portion of the obturator internus, and the superior surface of the levator ani and coccygeus muscles. That portion of the fascia which covers the superior surface of the pelvic diaphragm is termed the superior fascia of the pelvic diaphragm (fascia diaphragmatis pelvis superior).

From the line of origin of the superior fascia of the pelvic diaphragm, the pelvic fascia continues downward to cover the mesial surface of the lower portion of the obturator internus. This fascia, the obturator fascia, is attached to the tuberosity and ramus of the ischium and the inferior ramus of the pubis.

Along the line of union of the superior fascia of the pelvic diaphragm and the obturator fascia,

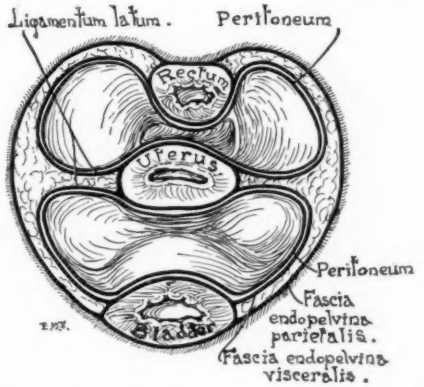

Fig. 5. Illustrating in a cross section made at a level of the corpus uteri, the distribution of the fascia endopelvina.

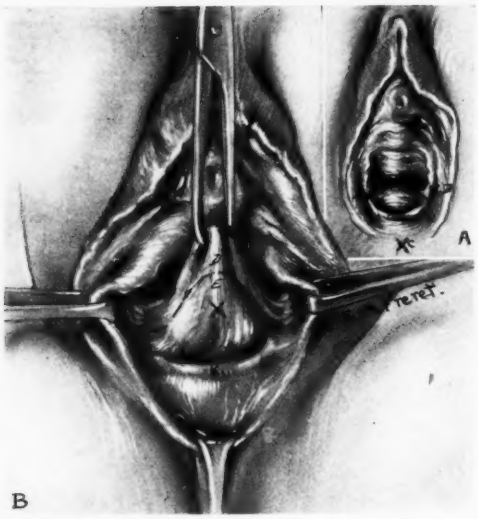

Fig. 6. The curved line *ADB* indicates the position of the initial incision in the rectovaginal septum, the uppermost point of which is 2 centimeters above the junction of the septum and the perineum.

there is a curved thickening in the pelvic fascia which extends from the posterior surface of the os pubis to the spine of the ischium, or in some instances to the iliopectineal line. This thickening is termed the white line (arcus tendineus musculi levatoris ani), and from it a large portion of the levator ani takes its origin.

From the white line, a thin layer of fascia extends over the inferior surface of the pelvic diaphragm, and is termed the inferior fascia of the pelvic diaphragm (fascia diaphragmatis pelvis inferior).

The superior and inferior fasciæ of the pelvic diaphragm form a compartment for the levator ani and coccygeus and both fuse along the anterior border of the levator ani with the superior layer of the urogenital trigone (triangular ligament). Posteriorly and mesially each of the two layers of the fascia of the pelvic diaphragm fuses in the sagittal plane with a corresponding layer from the opposite side.

In the above description of the pelvic fascia, it is apparent that the term "pelvic fascia" applies only to the fascial coverings of the pelvic muscles.

Fascia endopelvina. As the pelvic muscles are surrounded by layers of the pelvic fascia, so the pelvic viscera are surrounded by sheaths of a thin fascia, which, according to the descriptions in standard textbooks on anatomy, originate from a common curved thickening in the superior fascia of the pelvic diaphragm. This arcuate thickening extends from a point a little to the outer side of and slightly above the lower border of the symphysis pubis to the spine of the ischium, and is termed the arcus tendineus of the pelvic fascia. Upon the origin of the visceral layers of the pelvic fascia all textbooks agree. On the contrary, Halban (4), who has made a careful and convincing study of the fasciæ of the pelvis, disagrees not only with the descriptions in standard works on anatomy, but also with those in the writings of gynecologists.

Halban describes a layer of thin fascia which is situated between the parietal peritoneum and the fascial coverings of the abdominal muscles, and terms it the fascia endo-abdominalis (Fig. 1). This fascia extends over the entire abdominal parietes and thoracic diaphragm, sends layers to the abdominal viscera, and descends into the pelvis to become the fascia endopelvina (Fig. 2), which is composed of a parietal and visceral portion. Halban contends that the fascia endopelvina is a distinct fascia which is not a part of the fascial sheaths of the pelvic muscles. He emphasizes the fact that the parietal portion of the fascia endopelvina, though lying upon and attached to the muscle fascia by connective tissue, is distinctly separable from it. It is apparent, therefore, that he recognizes two distinct fasciæ in the pelvis, the muscle fascia and the fascia endopelvina, the distribution of which is clearly illustrated in the accompanying diagrams (Figs. 1, 2, 3, 4, and 5.)

To the various layers of the visceral portion of the fascia endopelvina are given definite names which are indicative of their situation. It is important to bear in mind that they are all parts of the same fascia and all fuse laterally.

In a consideration of cystocele and rectocele, the layers of the fascia endopelvina which are of greatest importance are the vesicovaginal layer, which lies between the bladder and the vagina, and the rectovaginal layer, which lies between the rectum and the vagina. It is to be noted that these layers fuse at the sides of the vagina to form a perivaginal sheath which is situated just without the vaginal musculature. Halban has given to this sheath the term "perivagina fibrosa."

Authors who have written upon the subjects of cystocele and rectocele have frequently used terms which are vague and misleading, among them the terms "fascia propria" of the vagina and rectum. These terms are poor ones in that they lead one to believe that there are individual fasciæ about the vagina and rectum without indicating their origins or situations. Halban's terms

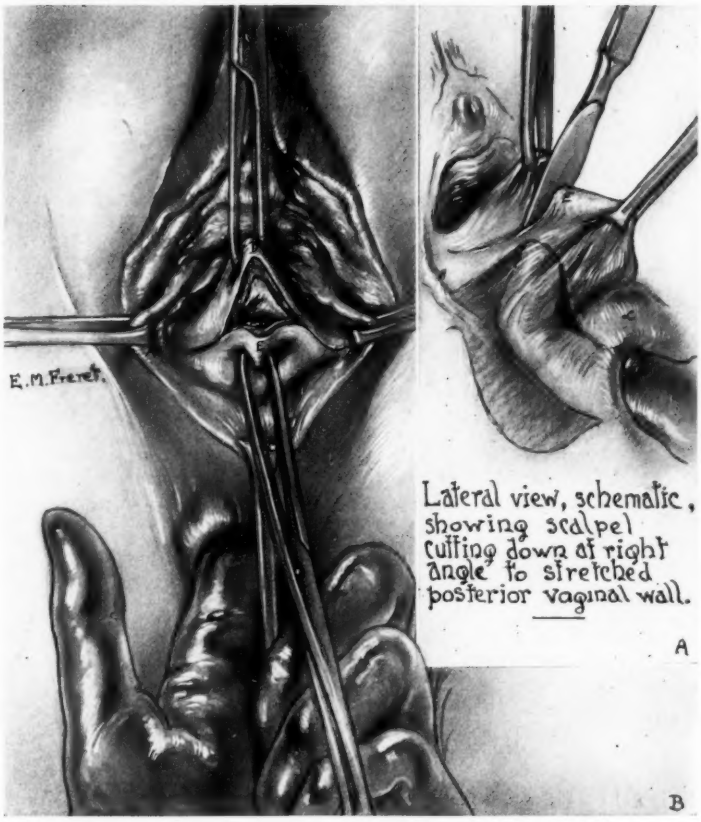

Fig. 7. Illustrating the method of making the initial incision, which passes through all layers of the septum and leads into the rectovaginal cleavage plane.

are definite and descriptive, and should be universally adopted.

The perineum. The term "perineum" is applied to that lozenge-shaped region at the pelvic outlet, which is bounded by the symphysis pubis, the coccyx, the greater sacrosciatic ligaments, the ischial tuberosities and rami, and the pubic rami. A straight line drawn between the anterior borders of the ischial tuberosities divides the region into an anterior urogenital triangle, and a posterior rectal triangle.

The urogenital triangle—to the gynecologist and obstetrician, the more important of the two perineal triangles—is bounded by the symphysis pubis anteriorly, by the rami of the pubes and ischia laterally, and by the posterior margins of both layers of the urogenital trigone posteriorly where they become continuous with each other and with the fascia of Colles.

Extending across the urogenital triangle, is a fascial plane, the urogenital trigone (triangular ligament), which forms a complete diaphragm over the anterior half of the perineum, except the urethral opening and the vaginal cleft. This important fascial structure is formed by the deep fascia of the perineum and is composed of two layers, the superior and inferior. Anteriorly both layers are attached to either edge of the inferior border of the pubis, laterally to the rami of the pubes and ischia, and posteriorly they unite and fuse with the deep layer of the superficial perineal fascia—the fascia of Colles. Laterally both layers of the trigone are continuous with the obturator fascia, and above the superior layer fuses at the

Fig. 8. The initial incision has been completed and the rectum has been separated from its abnormal attachments.

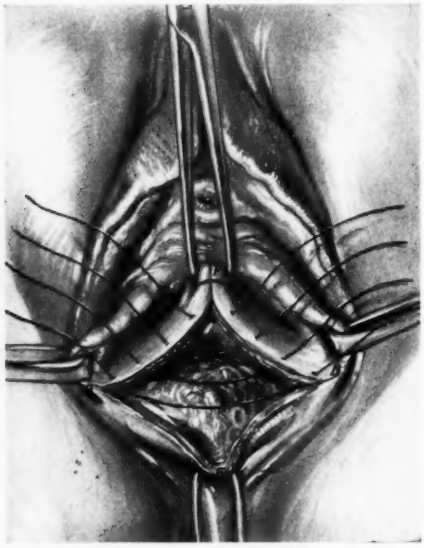

Fig. 10. Illustrating the approximation of the cut edges of the non-herniated portion of the rectovaginal septum, which is composed of the vaginal mucosa with its tunica propria, a circular muscular layer, a longitudinal muscular layer and the rectovaginal fascia.

Fig. 9. The excision of the herniated portion of the rectovaginal septum is adapted to the requirements of the individual case.

vaginal cleft with the fascia about the levator ani and with the fascia which surrounds the vagina, the fascia endopelvina. Along this line of fusion, the inferior layer of the trigone fuses with the superior layer. Robert Frank (3) called attention to these important facts in 1917.

The urogenital trigone in the female is perforated by the vagina, by the urethra, and by a small opening just behind the subpubic ligament through which passes the dorsal vein of the clitoris. Immediately behind the venous opening

Fig. 11. The denudation of the perineum is made with an Emmett scissors.

there is a thickened portion of the trigone termed the transverse ligament of the pubis.

Between the superior and inferior layers of the urogenital trigone there is a closed space, the deep perineal interspace. This space contains branches of the internal pudic vessels and nerves and a thin muscular sheet, which is composed of the deep transversus perinei muscle posteriorly and fibers which correspond to the compressor urethræ in the male anteriorly. The deep transversus perinei arises from the tuberosity of the ischium, passes behind the vagina, and inserts into the central tendon of the perineum. The anterior fibers of the muscular sheet are attached in the central tendon of the perineum and pass forward closely encircling the vagina and either surround the urethra or pass in front of it in the interval between urethra and transverse ligament. This muscular sheet has been termed the urogenital sphincter.

Over the urogenital triangle there is a superficial fascia, the superficial perineal fascia, which is composed of two layers, a superficial and a deep layer. The superficial layer, which is fat-filled, is continuous anteriorly and laterally with a corresponding layer on the abdominal wall and thighs. The deep layer, the fascia of Colles, which is membranous, is continuous anteriorly with Scarpa's fascia, is attached laterally to the rami of the pubes and ischia, and posteriorly blends with the urogenital trigone.

Between the inferior layer of the urogenital trigone and the fascia of Colles there is a tri-

Fig. 12. Suture *1* passes through the entire thickness of the perineum and includes the fascia about the levator ani where it is attached to the urogenital trigone. Sutures *2* and *3* pass through the same tissues.

angular space, the superficial perineal interspace, which contains the superficial perineal muscles, the long pudendal nerves, the glands of Bartholin, the crura of the clitoris, the vestibular bulb, the ischiocavernosus and bulbocavernosus muscles, and the superficial transversus perinei muscles attached to the ischial tuberosities and inserted into the central tendon of the perineum. In a practical consideration of the damaged pelvic floor, the superficial perineal muscles, except the transversus perinei and sphincter ani, are of little importance.

When we consider the anatomy of the perineum, it is important to distinguish between the perineum as described above and the perineal body, that triangular fibromuscular mass which separates the vagina from the rectum and anus.

Under a description of the urogenital triangle of the perineum the sphincter ani should be included.

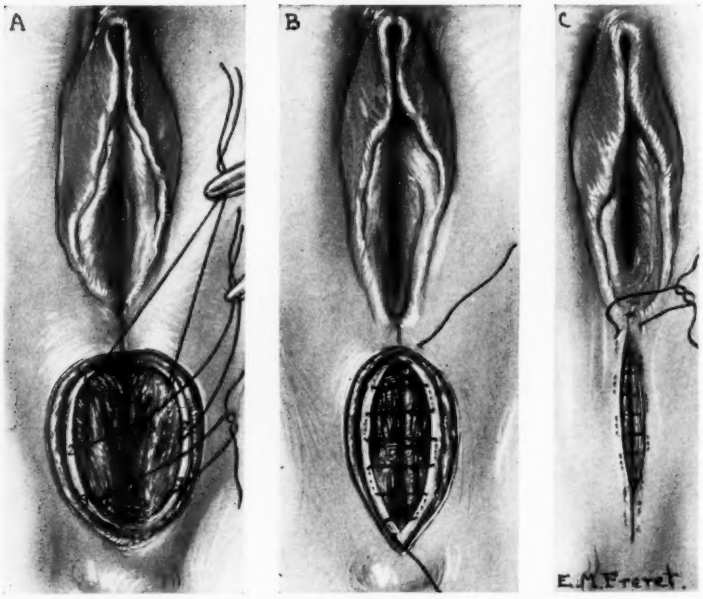

Fig. 13. After the deep sutures *1*, *2*, and *3* have been tied, the fascia of Colles is sutured as illustrated and the skin is closed by a subcutaneous suture.

This important muscle arises from the coccyx and from the ligamentum anococcygeum. It passes forward and around the anus to insert into the superficial perineal fascia and the central tendon of the perineum. Anteriorly its fibers may join those of the transversus perinei and sphincter aginæ. The central tendon of the perineum, which has been frequently mentioned, is located in the midline of the perineal body in front of the anus. It is the tendon of insertion for the following muscles: the sphincter ani, the transversus perinei, superficial and deep, and the sphincter vaginæ.

FUNCTION OF THE PELVIC MUSCLES AND FASCIÆ

The levator ani. In considering the function of the levator ani muscle it is important to remember that it is in reality composed of two muscles. The posterior portion of the muscle, the iliococcygeus, from the gynecological viewpoint, is relatively unimportant, as in woman its function is merely to bend the coccyx forward and to assist feebly the anterior portion of the muscle in its function. The pubococcygeus and puborectalis, the two groups of fibers which compose the anterior portion of the muscle, are the essentially important segments.

In commenting upon the function of the anterior portion of the levator, Robert Dickinson (2) has said: "I venture to affirm that there is no considerable muscle in the body the form and function of which are more difficult to understand than those of the levator ani, and about which such nebulous impressions prevail. . . . One commonly meets with the idea that the levator is a kind of muscular funnel tapering to the anus and serving to pull it directly upward after defæcation. This is absolutely untrue. The muscle rather resembles a horseshoe, a sling attached to the pubes in front, its sweep reaching horizontally backward, to encircle like a collar the rectum and vagina. Its action in woman is to drag the lower end of the vagina and rectum forward, level to the symphysis."

In a detailed description of the anatomy and physiology of the levator ani, Sturmdorf has clearly summarized its functions in the following statement: "Concisely stated, the levator ani diminishes the force of intra-abdominal pressure upon the pelvic contents by deflecting the direction of that pressure, augments the resistance to pressure by closing the uterovaginal angle, and obstructs the pelvic outlet against the pressure by compressing the vaginal canal. It is the tensor of the pelvic fascia, the antagonist of the diaphragm and

the abdominal muscles, contracting when these opposing muscles contract and relaxing when they relax. When intact, it maintains the equilibrium of the pelvic organs; when its integrity is impaired, equilibrium is disturbed."

Pelvic fascia. The pelvic fascia, as that term is here used, forms sheaths for the pelvic muscles, and through its attachments fixes them in their normal positions.

Fascia endopelvina. The function of the fascia endopelvina has been a subject about which there has been much controversy. Equally eminent authorities have held conflicting views concerning its importance. With others, Martin has contended that the pelvic organs are held in normal position principally by the fascia endopelvina, and attributes to the pelvic muscles a relatively unimportant rôle in their support. On the contrary, Halban and Tandler (5), who have made extensive studies of the subject, contend that the pelvic muscles, through their contractile force, are principally responsible for the support of the pelvic viscera, though they attribute to the fascia endopelvina an important rôle in the mechanism of pelvic support.

Regardless of either theory it is unreasonable to believe that the fascia supports the pelvic organs through sheer tensile strength, for it does not have sufficient strength to withstand the more or less constant strain of intra-abdominal pressure.

The more logical theory is that of Halban and Tandler in which the fascia endopelvina is considered as a fascial sheath, surrounding completely the uterus, bladder, and rectum, and fixing these structures in their normal positions, but allowing a normal degree of mobility. This fascial sheath plays a part in resisting the force of intra-abdominal pressure and gravity, but that part is relatively less important than that of the muscles of the pelvic diaphragm, which through their contractile force give the principal support to the pelvic viscera. It is obviously of the greatest importance, that in operating for the correction of uterine prolapse, cystocele, and rectocele, the surgeon keep clearly in mind this theory which does not recognize the fascia endopelvina and the pelvic muscles as mere anatomic buttresses against displacement of the pelvic organs, but which especially stresses the importance of the pelvic musculature in their support.

To the theory of Halban and Tandler there are antagonists who constantly cite, as evidence against the theory, cases in which there is marked levator impairment without descensus of the uterus. When analyzing such cases, one must not lose sight of the fact that such patients usually have a marked impairment of the abdominal muscles which play an important part in the production and maintenance of intra-abdominal pressure. The impairment in the force which these muscles usually exert is a result of pregnancy, and in most instances is of a degree sufficient to equalize the impairment in the function of the levator. The levator in such cases, though in a relatively inefficient condition functionally, is still able to maintain its influence in the support of the pelvic organs because of the reduced intra-abdominal pressure which it has to antagonize.

The perineum. The perineum, through the attachments of the urogenital trigone to the bony pelvis, constitutes an anchorage for the levator ani, the pelvic fascia, and the fascia endopelvina at the pelvic outlet. The superficial muscles of the perineum have as their chief function the fixation of the central tendon of the perineum.

CONDITIONS OF THE PELVIC FLOOR CAUSED BY PARTURITION

When the conditions of the pelvic floor which are caused by parturition are discussed, it is important to define clearly the meaning of the term "pelvic floor." In the present instance, it is desirable to have it include not only the musculofascial pelvic diaphragm, but also the rectovaginal septum and the perineum. It does not, however, include the vesicovaginal septum and the tissues which form the floor of the cul-de-sac of Douglas.

The conditions which result from injury to the pelvic floor at the time of parturition may be classified as follows: (1) general relaxation of the pelvic floor; (2) rectocele; (3) injuries of the levator ani and its fascia; (4) injuries of the perineum.

General relaxation of the pelvic floor. Every gynecologist is consulted by patients who complain of pelvic discomfort from a partial loss of normal tone in the vaginal septa, the levator ani, and the perineum, a condition which is a result of labor in individuals whose tissues do not involute properly, or of a prolonged second stage of labor in which the normal tissues have been subjected to pressure and distention without laceration. An analogy can be drawn between such a condition in the pelvic fasciæ and musculature, and that which is so frequently seen after pregnancy in the muscles and fasciæ of the abdominal wall. General relaxation of the pelvic floor is an entity with which the gynecologist has to deal and one which calls for operative correction in certain cases.

Rectocele. Rectocele may be defined as a hernial protrusion of the rectum into the vagina, always a result of injury to the rectovaginal septum at the time of parturition.

The injury of the septum which is the forerunner of a rectocele is not a constant one. In an occasional case it may occur at a point near the cervix, in other cases it may occur midvagina, but in the large majority of cases occurs at or near the junction of the rectovaginal septum with the urogenital trigone. The laceration may occur in the midline or in one or both vaginal sulci. Regardless of its situation, the resulting rectocele always protrudes symmetrically in the midline of the vagina.

An examination of a microscopic section from the normal rectovaginal septum reveals the fact that it consists of a mucous membrane composed of squamous epithelium with its tunica propria, a circular muscular layer, a longitudinal muscle layer outside of which there is the rectovaginal layer from the fascia endopelvina. If this arrangement of the tissues is kept in mind, the injury essential for the development of a rectocele is obviously one which involves the rectovaginal fascia, the fascial partition between the rectum and the vagina. Following the injury to the fascia and the retraction of the lacerated edges, intra-abdominal pressure forces the rectum into the hernial opening and gradually develops a hernial protrusion into the vagina.

In the course of the development of a rectocele, the rectal wall dilates and becomes abnormally attached in the sacculated vaginal wall. As the protrusion increases in size, nature makes an unsuccessful effort to correct or stop the development through a general hypertrophy throughout the vaginal mucosa and muscular layers. The marked thickening of the coverings of a large rectocele of long standing is apparent even in a gross examination of the tissues.

An inspection of Figure 2 convinces one of the possibility of vaginal lacerations which pass through all layers of the septum and through the rectovaginal fascia, with the subsequent development of a rectocele, but with no injury to the fascial covering of the pelvic diaphragm, and, therefore, no injury to the levator ani muscle. This is a point of importance, as it has been generally believed that sulcus injuries necessarily involve the levator and its fascia.

Injuries of the levator ani and its fascia. Textbooks on obstetrics usually contain vivid illustrations of lacerations in the levator ani resulting from spontaneous labor. Such illustrations are in most instances misleading, for the injuries which they depict are seldom, if ever, encountered by the most competent observers in the routine of obstetric practice. Studdiford (8), who made careful studies of the injuries to the pelvic floor due to childbirth, wrote, in 1915, as follows: "I have never been able to demonstrate in either recent or old lacerations of the pelvic floor, the rupture of the fibers of the pubococcygeus that is so graphically pictured in many textbooks and do not believe that it takes place except in badly executed forceps delivery." After an unusually extensive study of dissections of the levator in cadavers and living subjects, Sturmdorf holds a similar opinion.

The common injury to the levator ani and its fascia, which occurs in both spontaneous and operative deliveries, is one in which the levator fibers normally inserted into the perineal body, are lacerated in conjunction with the urogenital trigone usually in the midline of the body.

In poorly executed operative deliveries, however, the levator bellies may be actually lacerated either transversely or longitudinally near the origin of the muscle or at a point midway between origin and insertion. When such lacerations occur it is not uncommon to see fat from the ischiorectal space protrude through the rent in the muscle.

The remote result of injury to the levator is by no means constant. If the more usual midline injury has occurred, the muscle in its retracted position may become markedly hypertrophied as a result of an attempt, on the part of nature, to compensate for the impairment due to the injury. If, on the contrary, the muscle fibers have actually been lacerated transversely, the retracted portions may undergo an atrophy of a degree which will make difficult, if not impossible, a satisfactory reconstruction of the pelvic floor. In either case the levator is no longer able to perform its function adequately in the resistance of the intra-abdominal pressure and in the maintenance of pelvic support.

Injuries of the perineum. When an attempt is made to reconstruct the lacerated perineum, it is of the greatest importance to keep in mind, that, to the deep perineal fascia or the urogenital trigone, the layers of the fascia about the vagina, the levator ani, and the rectum, are attached. It is apparent, therefore, that any injury to this important fascia, will necessarily involve the structures for which it forms an attachment.

The perineum may be lacerated completely, or an incomplete laceration which reaches as far as the sphincter ani, may occur. It is with the latter class of injuries that the present article deals.

The site of the injury of the perineum is variable. All obstetricians are familiar with the simple straight midline laceration which passes through the median raphe of the perineum. This type of injury is notorious for the ease with which it heals and because levator impairment and rectocele

seldom follow it. The more serious type of injury is that which passes to one or both sides of the midline and which involves the perineal muscles and fascia, especially the lateral portion of the urogenital trigone. Such injuries, even when primary repair has been performed, often result in permanent damage to the structures involved.

The result of an injury to the lateral portion of the urogenital trigone may be a rectocele or diastasis of the levator or both. When such an injury occurs, the rectovaginal fascia is usually torn from its attachment to the trigone, and regardless of the care with which the repair is attempted a rectocele in the lower one-third of the vagina is very likely to result. Such an injury results, as a rule, in the lateral retraction of the injured portions of the trigone and the levatores which are attached to its superior layer. When retraction occurs, the levator cleft (hiatus genitalis) is converted from a V-shaped space to a U-shaped gap, and the anterior margins of the levator muscles become almost parallel to each other.

In view of these facts, it follows that the reconstruction of the damaged pelvic floor in which a rectocele has developed, consists of three distinct operative procedures: correction of rectocele, the restoration of the levator ani to normal position and function, and the reconstruction of perineum.

The operation herein illustrated consists of the following steps: (1) incision of the rectovaginal septum; (2) removal of the rectum from the herniated portion of the rectovaginal septum; (3) excision of the herniated portion of the rectovaginal septum; (4) closure of the hernial opening in the rectovaginal septum; (5) denudation of the perineum; (6) replacement of the levator ani and reconstruction of the perineum.

1. *Incision of the rectovaginal septum.* In practically all flap-splitting operations on the pelvic floor, the initial incision is made along or close to the junction of vaginal mucosa and the perineal skin. The flap is elevated either by scissors being plunged into the tissues, between the rectum and vagina or by a more cautious nibbling dissection with scalpel or scissors. Both methods are objectionable because they do not make for a clean-cut and accurate entrance into the natural cleavage plane which lies between the rectum and the rectovaginal fascia. Very frequently, when the scissors are plunged into the tissues, the natural cleavage plane is missed and a false plane of cleavage is produced with the result that a part of the septum or fascia remains on the rectum. The same error is also easily made in the more cautious dissection which unnecessarily traumatizes the tissues and causes an unnecessary blood loss.

The importance of accurately entering and following natural cleavage planes has been emphasized by Bissell (1), who, in an article, published in 1918, on a fascia-lapping operation for rectocele, stresses the fact that there is a natural cleavage plane between the rectum and rectovaginal fascia, which he terms a "cellular" area, along which the rectum is easily separated from its attachment to the fascia. At the same time he describes the method by which he reaches the cleavage plane as follows: "The mucous membrane of the perineum is first removed and the rectovaginal fascia exposed. This fascia is incised at about where it merges with the perineum or its remaining structures. When the fascia is incised, a cellular area is entered, which area constitutes the line of demarcation between rectum and vagina."

In the procedure herein described the primary incision is neither made at the mucocutaneous junction of the perineum nor at the junction of the septum and the perineum, but is placed at a point 2 centimeters above the junction of the septum and the perineum, because at this level there is little or no scar tissue encountered, and the rectum, which has been pulled downward while the septum has been elevated by clamps (Fig. 7), is barely attached to the underside of the rectovaginal fascia by a delicate areolar tissue. The entrance into the rectovaginal cleavage plane is, therefore, made with greater ease than when the incision is placed at a lower level where there is the greatest amount of scar tissue and where the rectum is more firmly adherent to the septum and perineum. The incision passes through all layers of the septum to the musculature of the rectum. As the scalpel passes through the septum, the cleavage plane is recognized by the presence of the delicate areolar tissue which attaches the rectum to the rectovaginal fascia.

2. *Removal of the rectum from the herniated portion of the rectovaginal septum.* When the initial incision in the rectovaginal septum is completed, the rectum is separated from its abnormal attachments by a finger covered with gauze. The separation is carried well beyond the area through which the rectum has protruded (Fig. 8).

3. *Excision of the herniated portion of the rectovaginal septum.* The outline of the excised portion of the rectovaginal septum will depend in each case upon the location and the extent of the hernial protrusion of the rectum into the vagina. In the case of a rectocele which protrudes through the lower one-third of the vagina, the removal of a triangular segment, as illustrated in Figure 9, will answer the purpose, while in higher rectoceles it

may be necessary to carry the lines of the excision almost parallel to each other from the initial incision to a point near the cervix. In any case the excision should be carried considerably beyond the herniated portion of the septum, so that the uninjured lateral portions of the septum may be used in the reconstruction.

4. *Closure of the hernial opening in the rectovaginal septum.* The hernial opening in the septum is closed by means of interrupted No. 2 chromic catgut sutures which include all layers of the rectovaginal septum, especial attention being given to the approximation of the musculofascial portion of the septum (Fig. 10).

5. *Denudation of the perineum.* When the rectovaginal septum has been closed to the level of the remains of the hymen, the perineum is denuded with the aid of an Emmett scissors as illustrated in Figure 11. Mucosa and skin only are removed. This denudation exposes the retracted portions of the perineum and prepares it for reconstruction.

6. *Replacement of the levator ani and reconstruction of the perineum.* Following the removal of the perineal skin and the vulvar mucosa the index finger of the operator's left hand is inserted into the space which is bounded by the newly reconstructed septum, the levator muscles, and the rectum. The rectum is pushed backward, and the line of attachment of the levator to the urogenital trigone is located. A No. 3 chromic catgut suture is then passed from point 1 (Fig. 12), just within the fascia of Colles, through the entire thickness of the perineum, and is made to include the fascial sheath of the levator just where it attaches to the trigone. The needle is then brought out between the halves of the perineum and is re-introduced and passed in a similar manner on the opposite side of the body. Two additional sutures are passed in exactly the same manner at points *2* and *3* (Fig. 12) and are made to include the same tissues as are included in suture *1*. In placing such sutures it is at times difficult to locate exactly the junction of the levator with the perineum, with the result that the sutures include small amounts of the levator fibers, a condition which will do no harm, as sutures so placed will not cause strangulation as do those placed about the bellies of the pubococcygei above the perineum. When the three sutures have been tied, the levator muscles have been drawn downward and toward the midline into a normal situation and relationship, without the production of any abnormal and objectionable rigid band of muscle between the rectum and the vagina. The muscles have been replaced by traction on their fascial sheaths. The normal V-shaped levator cleft has been restored.

In tying the sutures (*1*, *2*, and *3*) the retracted halves of the perineum have also been restored to a normal position and to a normal relationship with the associated fasciæ.

The fascia of Colles is closed by a continuous suture of No. 2 chromic catgut placed from above downward. When this suture has reached the lower angle of the perineal wound, it is carried upward as a subcuticular suture and tied at the top of the wound (Fig. 13).

In view of the fact that the present article is offered as a preliminary report, it is obviously undesirable to make any statistical statement at this time covering the end-results which have followed its application. It is safe, however, to state that the operation was devised in the summer of 1923 and was immediately employed, with success, in the correction of several extremely large rectoceles. Since that time it has been extensively used by nine members of the staff of the Woman's Hospital with consistently desirable results in the cure of rectocele and in the restoration of the levator ani muscle to normal position and function.

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A METHOD OF ASSISTING IN THE TREATMENT OF DISPROPORTION AND A MODIFIED METHOD FOR ESTIMATING THE RELATION OF THE FETAL HEAD TO THE PELVIS

By BETHEL SOLOMONS, M.D., F.R.C.P.I., Dublin, Ireland
Master of the Rotunda Hospital

SIMPLICITY is the keynote of success in obstetrics, and I have obtained much success at the Rotunda Hospital by the following simple treatment of some cases of disproportion.

The method is chiefly used when it has been determined by the Mueller-Kerr or by my own maneuver, that the head will fit in and be delivered by the natural passages. It is also used in cases of both primigravidæ and multiparæ when there is no fixation of the presenting part, and when natural delivery appears likely, *but* when every little help is indicated. It is of special assistance in patients in whom the inclination of the pelvis is greater than usual, i.e., the pubis is on a much lower level than the promontory. The head then tends to over-ride the pubis and to give a false impression of the amount of disproportion.

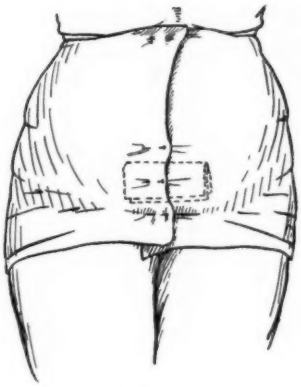

Fig. 2.

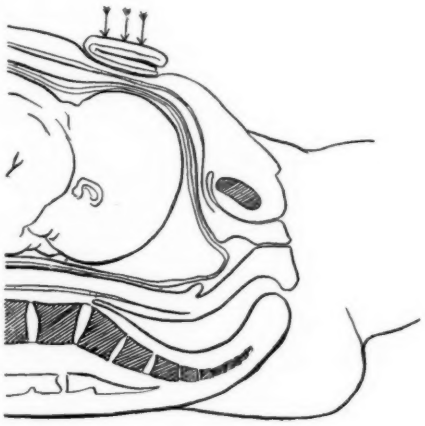

Fig. 1.

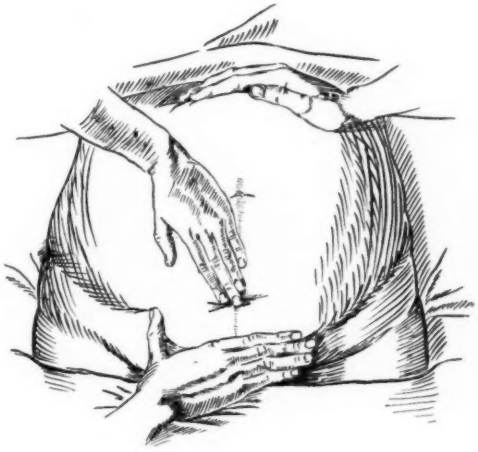

Fig. 3.

It should be used as a routine in a trial of labor. The method is as follows:

A pad of gamgee tissue is folded in 4 thicknesses, 11.5 cubic centimeters in width, by 21 cubic centimeters in length, the entire thickness being 4.5 centimeters (Fig. 1). The head is then put into the engaging position, getting as much flexion as possible. The pad is next applied. The operator holds the pad in position over the fetal head, while an assistant applies a tight abdominal binder which is pinned from above downward. Figure 2 shows the pad and binder in position.

When the abdomen is definitely pendulous, a pad should be applied in addition to the upper and pendulous part, to bring the fetus in the axis of the upper strait.

I have lately practiced the following method of estimating the relation of the head to the pelvis, which is a vast improvement on the original Mueller maneuver. The obstetrician sits on the right side of the patient and makes the Pawlik grip, while he places the fingers of his other hand on the fetal head and presses it downward in the axis of the superior strait. At the same time, an assistant pushes on the fetal breech from the fundus (Fig. 3). If this does not give definite information about conditions, the Mueller-Kerr modification is practiced.

NEW ANAL INSTRUMENTS

By W. D. PENNINGTON, M.D., Chicago

THE instruments described are (1) a tubular self-retaining dielectric cryptoscope; (2) a tubular self-retaining dielectric anoscope; and (3) a bivalve self-retaining adjustable anoscope. Each instrument is 3½ inches long. They are flanged at the proximal end and are conical in shape at the distal end so that they may be easily introduced. Each has a shoulder 2½ inches from the proximal end to prevent exshock, are especially adapted for use by those who treat diseases in and about anal regions with the electric current.

The cryptoscope is composed of the cryptoscope proper, an obturator, and an insulated detachable lamp, and it has a bridge across the fenestra extending three-fourths inch from the proximal end to prevent the sphincter from obstructing the view when it is desired to examine

Fig. 1. Tubular self-retaining dielectric cryptoscope.

Fig. 2. Tubular self-retaining dielectric anoscope.

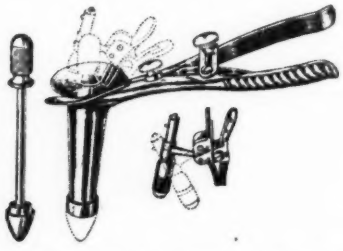

Fig. 3. Bivalve self-retaining adjustable anoscope.

pulsion, an assistant being thus unnecessary. The cryptoscope and anoscope are made in two diameters: three-fourths and one inch, while the anoscope is made in but one diameter, three-fourths inch.

Each instrument has a detachable insulated device, easily adjustable for illumination of the various fields in the region to be explored, examined, and treated. The insulation prevents shock in the event of a short circuit. The cryptoscope and anoscope are made of dielectric material and, as they prevent the possibility of and treat the crypts and other diseased conditions which may be exposed.

The anoscope is made up of three parts, the anoscope proper, an obturator, and a detachable and adjustable insulated lamp.

The bivalve anoscope has four parts. The anoscope proper, an obturator, an insulated and detachable adjustable lamp, and a slotted plate and thumb screw for adjustment.

The lamp device which is used in the instrument was invented by Dr. D. C. McKinney, of Buffalo.

EDITORIALS

SURGERY, GYNECOLOGY AND OBSTETRICS

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JUNE, 1928

SURGICAL PROCEDURES AND RADIUM IN THE TREATMENT OF CANCER OF THE MOUTH AND THROAT

THE use of radium in the treatment of cancer of the mouth and throat is a striking example of the misuse of a very valuable therapeutic agent.

Owing largely to the education of the public by the medical profession, many more patients with small chronic fissures or leucoplakia of the lips, or minor ulcerations of the tongue or throat, are presenting themselves for treatment fearing the possibility of cancer. One rarely sees, at the present time, a patient with extensive cancer of the mouth or throat who has not been under the care of a physician.

Of equal importance to the education of the public is the education of the medical profession to give the best possible treatment to these patients with early lesions of the mouth and throat.

Before the advent of radium, the patient with a suspicious ulcer of the mouth or throat presented himself to a surgeon who excised the lesion, either with knife or cautery, for microscopic diagnosis. If the lesion proved to be malignant, the gland-bearing fascia that drained the area was removed. If it proved to be benign, the patient was well-rid of the trouble and no further treatment was necessary.

Today such lesions are frequently treated with radium by the radiologist, the dermatologist, or the physician with a small amount of radium at his disposal. Following this, one of two things may occur. The local lesion clears up entirely, only to recur, however, in the lymph nodes of the neck in a few months or years, or the local lesion becomes more active and metastatic nodes appear in the neck. Frequently when these nodes appear, the patient has a tooth removed believing it to be the cause. Later the mass in the neck is explored and found to be an inoperable, metastatic carcinoma.

In cases of cancer of the larynx, removal of the growth by thyrotomy, with or without removal of the cartilage or laryngectomy, offers the patient the best chance of cure in the intrinsic case. Although radium has been used and is still being used in intrinsic cancer of the larynx, the end-results are not good and the sloughing of the cartilage which frequently follows is sometimes disastrous. Recently Portmann and Harmer recommended removal of the cartilage from both thyroid cartilages, leaving only a frame of cartilage in order to avoid secondary sloughing, and then using radium directly against the growth. Although their results indicate that this may be of use in inoperable cases, it should not be used if the lesion can be removed surgically.

In this country, radium is generally used in the treatment of extrinsic epitheliomata of

the larynx; operation is not attempted. Sir Wilfred Trotter of the University College Hospital, London, performs lateral pharyngotomy, which is a great advance in the surgical treatment of growths of the epiglottis, aryepiglottic folds and the postcricoid region. Trotter reported the results of his first work in 1913 and has since perfected the procedure. After removal of postcricoid tumors, he reconstructs the upper end of the œsophagus and pharynx with a flap turned in from the neck. In many of his cases the condition was entirely cleared up by surgical removal.

Radium has, however, a distinct place in the treatment of cancer of the mouth and throat but it is being used at the present time in the treatment of many lesions for which it is doing more harm than good. Patients with rapidly growing, extensive cancers of the tongue and antrum, who could not possibly be benefited by any surgical procedure have been entirely cured of their trouble by radium. Radium seems to be of help in pre-operative and post-operative treatment. It should not be used, however, to treat the questionable primary lesion, which should be excised for microscopic diagnosis, nor should it take the place of the removal of the gland-bearing fascia of the neck.

What has been accomplished by radium in the treatment of cancer of the mouth and throat has been overshadowed by the number of inoperable cases that have increased at least 25 per cent, as a result of its indiscriminate use in the treatment of the primary lesion, and of nodes of the neck. GORDON B. NEW.

PRACTICE OF OTOLOGY IN THE SUMMER MONTHS

THE practice of otology in the summer months varies somewhat from that in other months. One difference is ascribable to swimming which produces an otitis media, especially in poorly cared for hotel pools that are used by a large number of house guests and by the enthusiasts of the neighborhood. In our large cities, particularly those on inland lakes, most of the public beaches are located in such a way that the shore projects at each end of the beach and tends to form a pocket which prevents to some extent the flow of water in this area. Further, the lavatories are often not conveniently located, on some beaches there are none available, so that pollution of the water by bathers who frequently stay on the beach for several hours during the hot days is encouraged.

Otitis media from swimming is caused by the entrance of polluted water into the nose and throat. This, followed by choking or blowing the nose hard, forces the infected water up the eustachian tubes to the middle ear. From experience in dispensary and private practice, it seems that otitis media of swimming origin, as contrasted with that from acute head colds in winter time, is definitely less severe and responds more quickly to early paracentesis and relatively infrequently continues to the stage of surgical mastoiditis.

It hardly seems necessary now to suggest that in cases showing definite signs and symptoms of surgical mastoiditis, there are several advantages in not operating at the onset of mastoid pain and tenderness. In the first place, it is not unusual to find that after delaying a few days, the mastoid symptoms disappear and recovery is complete without operation. Secondly, especially is it true that a child may have mastoid pain and tenderness from conditions other than surgical mastoiditis. Lastly, in unquestionable cases of acute surgical mastoiditis, a delay of a few days allows the surrounding tissue and the general resistance to the offending organism to increase greatly. Delayed mastoidectomy in cases of uncomplicated acute mastoiditis, unquestionably shortens the postoperative course.

One symptom which has been found very significant in questionable cases, is night pain in the affected mastoid. It is especially helpful in children, who frequently have a complicated assortment of signs and symptoms.

Parenthetically, surgical mastoidectomy as performed by well trained American otologists is not surpassed by our European confrères. A visit to the larger centers of Europe during the past spring, revealed that our surgical work has a few definite advantages. First, our work is less radical, yet we do explore when exploration is indicated, rather than as a matter of routine. Second, we think of the postoperative appearance of our wound and therefore partially close the wound at operation, which lessens the scar and greatly shortens the postoperative course of treatment and healing. Third, we are infinitely more mindful that our patient shall not endure unnecessary pain and that he has a definite personal pride and modesty that should be respected.

During the past two summers the great increase in foreign travel has produced some unusual ear manifestations, especially in infection of the external auditory canal due to the fungus aspergillus niger. Two cases observed were in patients who just returned from extensive travel, one spending 7 months on a world cruise, the other 3 months in the interior of China. A third was found in a man who spends much time traveling in this country. The diagnosis was made from a microscopic examination of the black mass, in which mycelia were found. In each case the patient sought relief from cerumen and returned with the suggestive black, thick secretion involving a small area of the canal membrane.

Furunculosis of the canal is also a definite feature of otology in the summer months, yet it does not seem to give evidence of any peculiar seasonal variations.

Examination of the deaf shows a marked increase during the warm months. In searching for a reason it seems that possibly the out-of-door life at this time of year impresses the deaf with their great limitations and so encourages them to seek relief. Also, the education of parents and children by the investigation of school children by otologists and the publicity in recent years of our otological societies, and their committees has greatly encouraged examination. The mechanical devices for the aid of deafness show a marked improvement, which is encouraging in that further refinement will develop as more demand is expressed for this means of relief.

The correction of the mild conduction type of deafness, constant or intermittent, as found in children, due to a partial closure of the eustachian tube from adenoids or mild nasal abnormalities, is a striking seasonal feature. Not infrequently, inattentive and scholastically poor students are completely changed by relief of their deafness, due to treatment of the eustachian tube or indirectly by correcting nose and throat conditions.

HORACE R. LYONS.

MASTER SURGEONS OF AMERICA

LOUIS SAMUEL McMURTRY

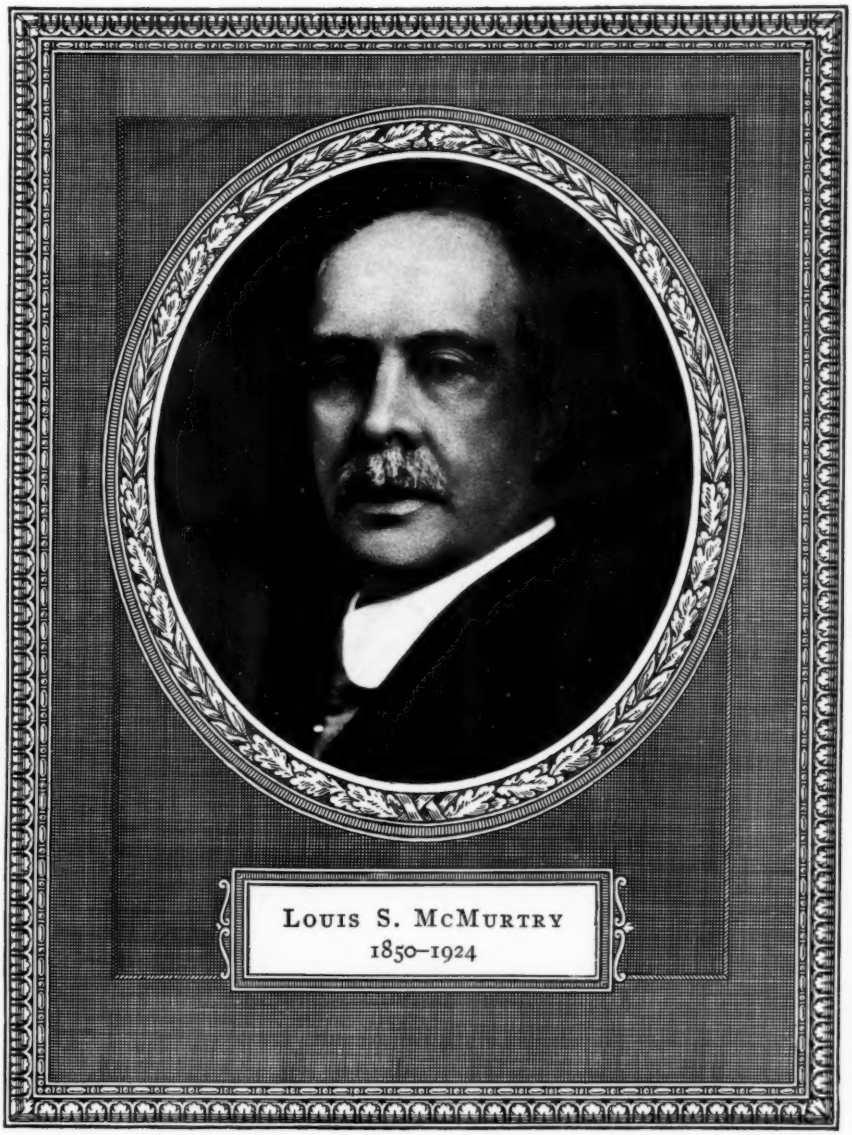
BORN in Harrodsburg, the seat of the first capital of Kentucky on September 14, 1850, of Scotch descent, Louis Samuel McMurtry was reared and educated in the Blue Grass, the aristocratic section of the State, and died in Louisville, the seat of his labors for over thirty-five years, on February 1, 1924.

Danville was long the cultural center of the State and it was here that young McMurtry obtained his academic education, receiving his degree in 1870. His alma mater, Centre College, numbered many distinguished names among its alumni. Unquestionably the atmosphere in which he at this time lived must have fired his ambitions and desires and have played no small part in determining him not only to study medicine, but to follow in his professional career along the same lines as the immortal McDowell.

At Danville, McMurtry came early, most likely during his academic studies, under the influence of Dr. John D. Jackson, a scholar, a student, a gentleman, and a philosopher, as well as a surgeon of distinction. He read medicine with Jackson for a year or two, dissecting the human body and becoming well grounded in anatomy before entering medical college.

Is it to be wondered at that he was enthusiastic in his studies or that he graduated after his course at Tulane with the honors of his class? His graduation in 1873 was followed by an interneship at the old Charité where he was chief medical resident. During this time he presented several papers bearing upon important medical topics. His impress upon his professors during these student days was so great that he was invited to make the address to the graduating class 2 years later.

Returning after his graduation to Danville he took up the active practice of medicine and surgery. With a year's intermission, during which time he taught anatomy in the University of Louisville, he continued his practice in the community until he moved to Louisville in 1888. These were busy years. Although the demand on his time in general practice was very great, he continued a diligent student of surgery and was one of the early converts to Listerism having been a student of the work of Pasteur. He was a life long admirer of the great French scientist. During this period his surgery was not confined, as later, to abdominal and pelvic work as may be noted by his papers which include the report of a successful ligation for aneurism of the subclavian artery and papers on general surgery.



LOUIS S. MCMURTRY
1850–1924

As stated earlier in this sketch he had been much impressed with the work of McDowell and determined to confine his work to abdominal and gynecological surgery which had become recognized as a distinct specialty as a result of Lawson Tait's studies in pathology. He therefore made a tour to Birmingham and became a student of this great and incomparable master. Perceiving on his return to his native State that his work demanded a wider field of usefulness he removed to Louisville. The demands upon him were great, his reputation as a surgeon had already been made. He was at this time widely and favorably known and his state medical association honored itself and him by electing him as its president in 1888.

It was about this time in his professional career that McMurtry became the intimate of Howard Kelly and Joseph Price. With these two he was most active in society work and in the promulgation of the newer concepts of the pathology of the female pelvis and its treatment and in preaching the doctrine of aseptic surgery. His reputation became international and he took a very prominent part in medical societies, his discussions being at all times forceful, logical, and convincing. The early advances in American surgery, its rapid strides, and the marvelous development of abdominal and general surgery was in a large measure due to these pioneers, all of them brilliant operators and teachers, blazing the trail which we now so easily tread.

McMurtry had become internationally known 10 years before this period as a result of his labors not only in establishing the claims of McDowell to priority in the field of ovariotomy but also in taking up the work of his preceptor, Dr. Jackson, in fostering the erection of a memorial to the great McDowell.

In 1890, McMurtry was called to the chair of abdominal surgery and gynecology in the Hospital College of Medicine, which chair he filled with distinction, being shortly made President of the faculty and occupying these appointments in the school until it was merged with the University of Louisville in 1910. He was most active in bringing about the merger of the medical colleges in Louisville and became president of the Medical Department of the University of Louisville remaining as such until the time of his death.

Of most genial disposition he was quick to resent what he deemed a wrong or an injustice. A friend of the young striving beginner, he demanded merit and hard work. Cultured and courtly in manner he was a raconteur of the rarest order and loved nothing better than association with congenial fellowmen. He was, as all great men are, a lover of good literature and of the poets. His life and work was in itself a poem, his operations were epics. He was a brilliant conversationalist and his travels and reading made him a most interesting companion.

He was particular in dress as he was in choice of language and because of his charming manner he was often called upon not only to preside at meetings but to make addresses, public and professional.

As a surgeon of keenest judgment, as an operator of brilliant and marvelous technique, as a diagnostician of wonderful acumen, insight, and sound reasoning, McMurtry had no superior and few equals, certainly in pelvic surgery. His early work with that of Price and Kelly has left its mark upon American surgery of today.

Doctor McMurtry in addition to his membership in the American Medical Association of which he was president in 1905–1906 was an active member of his state and county societies, a fellow of the British Gynæcological Society and of the Edinburgh Society of Obstetricians, and one of the founders of the American Society of Obstetricians and Gynecologists, of the Southern Surgical Association, and also of the American College of Surgeons in which he took a most active interest. He was a member of the American Surgical Association and at the time of his death was president of the Kentucky State Board of Health which post he had held since 1919.

During the great World War, he organized the first unit in Kentucky that saw oversea duty. He was appointed by the Surgeon General as Military Aid to the Governor which post he filled until its duties became too onerous.

His illness was a short one, death being due to pneumonia of four days' duration. When the time came to join the innumerable caravan he went,

"Like one that draws the drapery of his couch
about him and lies down to pleasant dreams."

LOUIS FRANK.

pag 448

a. Facies superior
b. Facies inferior
c. Tertium internodium
d. Principium secundi internodij
e. Secundi internodij osficula fracta
f. Os Sesamoidum
g. Insertio tendinis in tertium internodium
h. Tendinis longitudo duodecim digitorum
i. Tendinis latitudo
k. Finis ipsius tendinis, quâ è ventre sui Musculi evulsus est.

PETRI
DE
MARCHETTIS
PATAVINI,
EQUITIS D. MARCI
& in Patrio Gymnasio Chirurgiæ olim,
nunc verò Anatomes Professoris

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THE SURGEON'S LIBRARY

OLD MASTERPIECES IN SURGERY

By ALFRED BROWN, M.D., F.A.C.S., Omaha

THE MEDICOSURGICAL OBSERVATIONS OF PIERRE MARCHETTI

AMONG the numerous surgical "Observations" published during the seventeenth century, the little book of Petri de Marchettis or Pierre Marchetti takes high rank, for though small in compass physically, it holds between its covers an epitome of the advance that surgery had made during the preceding century. Marchetti was born in 1580, just as Paré was passing off the surgical stage, and lived until 1673. A native of Padua, he became first professor of surgery and later professor of anatomy at the University, where he was distinguished as a fearless and clever operator.

In 1664 his "Observations" appeared, according to Haller, sixty-eight in number, but the volume illustrated here was published in 1665 and contains but sixty-three. Following these are small books entitled (a) "Concerning Fistulas of the Anus and Rectum," (b) "Concerning Ulcers and Fistulas of the Urethra," and (c) "Concerning Spina Ventosa." The book is entitled "A syllogism of the rare medicosurgical observations of Petri de Marchettis of Padua, Knight of St. Mark, formerly professor of surgery and now professor of anatomy in his native University; Amsterdam, from the work-shop of Petri le Grand, 1665."

The great interest displayed in wounds of the head is particularly prominent in all the surgeries of the sixteenth and seventeenth centuries. Other forms of operative work like the stone and hernia were left to the barber surgeons and charlatans, but for some reason or other these head wounds were considered in the domain of the educated surgeon. In all parts of Europe there was constant warfare and war meant hand to hand fighting, so that clubs came into use and broken heads were common. Thus material for observation and treatment was abundant but does not seem to have been used for scientific purposes. The surgeon advised trephining, and if he wrote a book or treatise described his instruments and technique, after which he considered his work done. From Brunschwig on there are illustrations of trephines and saws, the prognosis and differentiation between concussion and compression are hinted at, as by Berengarius, but of detailed examination with the findings there is little said.

Marchetti in his clinical observation excelled his predecessors. A report of one of his cases reads almost like one of the present day; and, while in many of these old histories, diagnosis is difficult, Marchetti places his material before the reader so carefully that a diagnosis can be hazarded with pretty fair confidence that it is correct. If he drew any conclusions from his clinical data he did not publish them, or make any attempt to establish the relationship between cause and effect. This is seen particularly in his Case V, a brain case, in which, by his description he uncovers the key to cerebral localization, but then stops and says nothing more. He describes the case as a large wound of the right side of the head in which a small piece of bone was driven in through the membranes into the brain "and on this account, above the other symptoms common to these wounds, this was the symptom proper to this (wound). Paralysis of the opposite arm and tongue, so that he was able neither to speak, nor move the right arm." For 20 days Marchetti tried all sorts of treatment in the way of dressing and ointments without avail. He then decided that a piece of bone was present "which having been grasped by a volsellum I drew out; to it was attached a portion of the pia membrane and of the brain, whence hardly half an hour later he recovered the motion of the tongue and arm." Several other brain cases are described with equal care; one of particular interest in which traumatic epilepsy was cured by removal of a piece of bone. He believes that unless the signs of compression are present, cranial wounds are not particularly dangerous. This portion of the work reveals the author as a surgeon of excellent judgment and it is a disappointment to find later that Marchetti believes in the Galenic doctrine that nerves and tendons should not be sutured because of the vital fluid that flows through them and attacks Severino for advocating suture of these structures. He says: "Indeed I have seen nerves or tendons unguardedly sutured by certain Barbers followed by convulsions and the death of the patient." He publishes only one illustration which represents a thumb bitten off by a horse, with avulsion of the flexor tendon at its junction with the belly of the muscle. It is a little difficult to understand why this case should have been singled out for illustration.

Marchetti's conclusions about penetrating wounds of the chest are remarkable in that he lets them alone unless indications for active interference arise. He operates upon patients suffering with empyema so his expectant treatment is dictated by judgment and not by fear.

REVIEWS OF NEW BOOKS IN SURGERY

THE reviewer must plead guilty of profound skepticism when reading current texts and treatises of surgery, since libraries contain innumerable single volumes and brief systems dealing with surgery and its operations. It would seem that the material and its exposition must be exhausted and yet books continue to appear and we are reliably informed by the publishing houses that more are to follow. Truly surgery has made great strides in the past two decades and must continue to advance to meet the ever changing demands of the biologic and pathologic responses of a fickle human race. So long as revisions and new works keep abreast with the time or better still add to or disseminate the newer thoughts which have had thorough investigation and trial, then there is a place for them, but simply a re-hash of the old can not be countenanced; nor can a superficial, vague, and indefinite delineation be accepted. Texts must keep abreast with the times and must weed out the obsolete.

THE seventh edition of volume ii of *The Operations of Surgery* by Rowlands and Turner[1] is before us. This volume is devoted entirely to the abdomen. A careful study of this book indicates that it is not, strictly speaking, a text of operative surgery but rather a text of the treatment of surgical diseases. The approach to the subject material is pleasing and rather unique in that the disease is briefly discussed from the standpoint of the etiology, symptoms, and diagnosis of a specific pathological process; the description of the pathology leads in a most natural manner to its treatment. Instead of recommending resection of the stomach for carcinoma, the authors discuss carcinoma of the stomach as seen at the operating table, taking into consideration such factors as secondary growths in the viscera, peritoneal invasion, and the site, size, and degree of fixation of the growth. Upon these factors depend the type and extent of operation, if an operation is indicated. The operations are painstakingly described, innumerable details so essential to success being included. This scheme of description is most lucid and makes for a clear understanding of the treatment of a specific condition. Many excellent illustrations further clarify the text. The authors have shown wisdom in their selection of salient points in the general discussion of diseases. The amount of information given in so few words is remarkable.

A careful review reveals only one omission of note. In the chapter describing the treatment of postoperative complications, massive collapse of the lung is not mentioned. The bizarre onset of this condition and not infrequently the dramatic relief which is afforded by means of posture is well deserving of mention.

J. A. WOLFER.

THE first volume of Lewis' *Practice of Surgery*[2] to come from the press is number 2. This system is to consist of 10 volumes, covering the entire field of surgery and is to be prepared by a large number of contributors, each having given especial attention to his subject. The science of surgery has advanced so rapidly in the past decade or two that many so-called general surgeons specialize in certain branches. This is more or less of a necessity due to the tremendous development of the various branches of surgery, including the physiological, biochemical, and physical aspects. A good illustration of such specialization is the Registry of Bone Sarcoma under the guidance of the American College of Surgeons and the intensive study of such material by Kolodny, his recent monograph being a monumental achievement for American surgery. There is no question but that such specially equipped men are well prepared to present their topics because of their intensive study of the clinical and experimental side as well as the literary aspect of their topic.

A number of years have elapsed since such an extensive work has been attempted, and it would seem timely that such a system should be prepared for the medical profession, especially when it is edited by a man of the scientific attainments of Dean Lewis.

This, the first volume to appear, is devoted to bones and joints. There is an excellent chapter on bone tumors. This brings to the reader in a comparatively brief and lucid manner the subject as it stands today. Osteomyelitis is presented in a very striking and detailed fashion. The reviewer has never seen so complete and orderly an exposition of the subject, correlating pathology, symptoms, and treatment. Injuries and diseases of the joints are equally well presented. Probably nowhere in any language can so thorough a delineation of the subject be found. This applies especially to bursitis, a condition of which so little is known by the average medical man. These chapters are most exhaustive.

A casual survey of the chapter on fractures would lead one to believe that the subject was inadequately covered, yet Cotton in his characteristic manner, backed by his tremenduous experience and keen acumen, in such space as is allotted to him, hits the mark very well.

This volume speaks well for the editor and his contributors and indicates that soon a most comprehensive and thoroughly modern system of

[1] THE OPERATIONS OF SURGERY. By R. P. Rowlands, O.B.E., M.S (Lond.). and Philip Turner, D.Sc., M.S. (Lond.), F.R.C.S. (Eng.), 7th ed., vol. 2.—The Abdomen. New York: The Macmillan Company, 1927.

[2] PRACTICE OF SURGERY. Edited by Dean Lewis, M.D., Sc.D. Hagerstown, Maryland: W. F. Prior Company, Inc., 1927.

surgery will be available for the medical profession. The volume is loose leaf so that material may be replaced from time to time as advance is made in the art and science of surgery. J. A. WOLFER.

THE first edition of *Emergencies of a General Practice*,[1] which was published in 1918 by the late N. C. Morse, has been revised and rewritten by Amos Watson Colcord. It deals with "the class of work in which the attending physician is called on to think quickly and to act with rapidity and good judgment." The thirteen chapters contain much interesting information, and the description of methods which the writers have found best. The subjects considered include removal of foreign bodies, first aid, treatment of asphyxiation, "surgical emergencies," medical emergencies, fractures and dislocations, amputations, obstetric emergencies, and various types of poisonings.

The propriety is questionable of including in a book of this type the brief descriptions of such surgical procedures as gut resections, treatment of ruptures of liver, spleen, and pancreas, of acute appendicitis, of ruptured ectopic pregnancy, of strangulated hernia, cæsarean section and the wiring of teeth for fractured mandible.

On page 120, F-561 shows a proctoclysis outfit which is captioned "Meinecke's hypodermoclysis apparatus." On page 127, Colcord says that "no doubt the permitting of first aid classes to dress wounds has been productive of much wound infection." He found that one-third of this infection had been first treated with iodine.

The writer seldom boils his instruments but is content to sterilize them in "denatured alcohol slightly over-neutralized with caustic soda." In his opinion a general anæsthetic is nearly always necessary in the removal of needles. In his discussion of poison ivy (an emergency?) the writer makes no mention of the antigen treatments.

Although this volume is interesting, it is a far cry from Lejar. FREDERICK CHRISTOPHER.

IN a very excellent monograph,[2] Fitzwilliams has presented the modern surgery of the tongue. It is based upon a wealth of material from the London hospitals and the literature. The chapter on macroglossia and fissures of the tongue is full of interest and very complete. Other chapters include injuries, inflammations, leucoplakia, syphilis, tuberculosis, and actinomycosis of the tongue. There is a discussion of submaxillary calculus, ranula, and thyroglossal tumors and cysts.

Of greatest value is the one hundred pages devoted to cancer of the tongue. In his preface, the author says that "cancer of the tongue, in place of being the hopeless disease that the laity, and many physicians, and some surgeons seem to think it is, is just as curable in its early stages as is cancer elsewhere. It is a duty therefore for each medical man to instruct the public as to the curability of cancer when recognized and treated in early stages. . . ." The clinical characteristics of the hard edged ulcer and the papillomatous excresence are clearly brought out.

The author favors surgical methods, which he fully describes, but includes an account of treatment by diathermy, radium, X-rays, and various other methods.

The text is clearly written and made more vivid by the many illustrative cases. It is a book full of interest to every medical man and may heartily be recommended. FREDERICK CHRISTOPHER.

DR. SLUDER has limited himself in *Nasal Neurology, Headaches and Eye Disorders*[3] to a description of nasal diseases which have as symptoms headaches, eye disorders, neurological phenomena of nasal origin controlled by nasal treatment, and neurological phenomena apparently not of nasal origin, but controlled by nasal treatment; and orbital abscess. There is here epitomized numerous articles he has published on rhinological problems with the hope of securing the interest and co-operation of ophthalmologists, neurologists, and internists on obscure problems connected with rhinology.

A clear idea of some of the fundamental facts on which Dr. Sluder's deductions are based is given in the introductory chapter by Dr. Jonathan Wright on the minute pathological anatomy of changes observed in nasal tissue chiefly from material from Dr. Sluder's cases.

The chapter on vacuum frontal headaches is of great interest. Dr. Sluder distinguishes two types, one a symptom of a disease which is easily recognizable and limited by the duration of the disease; the other arising in some obscure condition of which the headache is the only symptom. It is with this headache that he is chiefly concerned and it is in such cases he believes the rhinologist can help. Such headaches are relieved by cocaine; permanent or long continued relief can come from astringents, but removal of the anterior part of the middle turbinates may be required.

About 50 pages are given to a detailed account of the syndrome of nasal ganglion neurosis. The description of the sphenopalatine ganglion with its anatomical relationship is full and clear. The ganglion is easily reached, since it is very constantly placed above the posterior tip of the middle turbinate and is separated from the nasal cavity by a small amount of areolar tissue and the nasal mucous membrane.

The discussion on hyperplastic sphenoiditis which extends over 100 pages may be read with advantage.

[1] EMERGENCIES OF A GENERAL PRACTICE. By the late Nathan Clark Morse, A.B., M.D., F.A.C.S. Revised and rewritten by Amos Watson Colcord, M.D. 2d. ed. St. Louis: C. V. Mosby Co., 1927.

[2] THE TONGUE AND ITS DISEASES. By Duncan C. L. Fitzwilliams, C.M.G., M.D., Ch.M., F.R.C.S. (Edin. and Eng). New York and London: Oxford University Press, 1927.

[3] NASAL NEUROLOGY, HEADACHES AND EYE DISORDERS. By Greenfield Sluder, M.D., F.A.C.S. St. Louis: The C. V. Mosby Co., 1927.

One is not amazed that sensory phenomena can arise from disease in it, but that they are uncommon in the experience of many competent neurologists. The question may well be asked: Are such disturbances unrecognized?

The relation of the Vidian nerve to the base of the cell is the cause of many of the syndromes, especially those with widespread pain extending from the mastoid to the arm and hand. Operation in such cases is by no means devoid of danger and Dr. Sluder insists that it be not undertaken without a thorough knowledge of the anatomy of the parts and a considerable degree of surgical skill. This chapter concludes with a description of the author's technique.

The chapter on neurological problems in rhinology deals to a large extent with phenomena not easily explained. Many are referable to reflex phenomena associated with the fifth nerve, others involve diverse opinions regarding the rôle of the sympathetic. This interesting chapter must be read in its entirety but until we know more about the sympathetic nervous system our belief will be more speculative than real.

The work is the result of years of study by a painstaking and indefatigable worker in rhinology and is a notable contribution to medical literature.

J. Gordon Wilson.

IT is very difficult to make a definition of minor surgery. All minor surgery is potentially major surgery, and often a distinction cannot be made. Not infrequently more judgment and more dexterity is required for this surgery than is needed for the standardized appendectomy or herniotomy.

In *Petite Chirurgie*[1] the writer has collected a veritable encyclopædia of information of interest to surgeons, practitioners, students, and nurses, but which far exceeds the limits which, to the reviewer, should restrict the field of minor surgery.

The first part of nine chapters includes the nursing care of the bed patient, the use of inhalations, vesicants, various types of enemata, the administration of hypodermics and intravenous medication, vaccination and serotherapy, the treatment of syncope, physiotherapy, pre-operative and postoperative treatment with attention to lavage catheterization, etc. The second part of six chapters takes up the accessories to an operation. Methods of sterilization, preparation of the hands, the operating table, and instruments and the types of anæsthesia. Minor surgical incisions, sutures and their preparation, cauterization, hæmostasis, and the treatment of accidental wounds are discussed.

The third part is devoted to dressings and bandages. The materials of dressings and the technique of their use is coupled with an excellent chapter on antiseptics. Bandages, slings, and trusses are thoroughly considered. The fourth part contains six chapters, devoted to the apparatus and physiotherapy employed in the treatment of fractures. The description of the paraphernalia for traction and suspensions seems inadequate. The one hundred and twenty pages given to the treatment of special fractures comprise an excellent compend of this subject, although fractures of the femur and spine scarcely have place in a minor surgery and one misses depiction of skeletal traction. Part five deals with the joints and includes sprains, joint effusions, dislocations, and some account of orthopedic treatment. The sixth part includes the lesser gynecological procedures, and catheterization. Part seven takes up the lesser surgery of the eye, nose, ears, larynx, and mouth.

The volume represents a great amount of serious effort and contains a mine of valuable information. Certain omissions and scope which exceeds the title do not prevent the reviewer from according it heartiest praise and commendation.

Frederick Christopher.

IN his monograph[2] of 118 pages, Roche begins with a historical review of pyelography, followed by a detailed discussion of the various solutions which have been used in this work from the beginning to date.

The matter of technique is given in detail, attention being centered on the dangers which may arise from its faulty execution. To avoid the possibility of courting danger, the author is careful as regards the selection of cases for pyelography. So decidedly opposed is he to pyelography as a routine procedure, that he states emphatically that it should never be resorted to except when a diagnosis cannot be arrived at by other means.

The various reactions following pyelography are discussed, as well as the different methods for their prevention. Indications and contra-indications are duly considered.

A very good detailed description of the normal pyelogram is given. The author wisely calls attention to the great difficulties at times encountered in the reading of pyelograms.

Descriptions of the various pyelograms as found in the presence of stone, tumor, tuberculosis, inflammatory conditions, etc., a discussion of the value of pyelography in the differential diagnosis of obscure abdominal pain, and of the value of pyelography in the presence of shadows in the X-rays plate and the relationship between obscure abdominal tumor and the kidney pelvis and ureter, are given, the author's statements carrying weight on account of his excellent judgment and his commendable conservatism.

The book is written with commendable clearness and will be a great help to all who wish to familiarize themselves with the technique of indications for pyelography and the dangers which may follow its use.

H. L. Kretschmer.

[1] Petite Chirurgie. By J. Maisonnet, Professor at Val de Grace. Paris: Gaston Doin et Cie, 1928.

[2] Pyelography; Its History, Technique, Uses and Dangers. By Alex. E. Roche, M.A., M.D., M.Ch. (Camb.), F.R.C.S. (Eng.). With an introduction by Sir John Thomson-Walker, O.B.E., M.B., C.M., F.R.C.S. New York: William Wood & Company, 1927.

PAPIN, surgeon of St. Joseph's Hospital in Paris, has just completed a beautiful two-volume work[1] on surgery of the kidney. The author has brought the literature on each subject up to date. However, it should be stated that the bibliography is almost entirely French, with some mention of German and less mention of English and American workers. The normal anatomy of the kidney, gross, topographical, and microscopic, is given many pages and is illustrated with colored plates; special attention is given to the kidney pelvis, calyces, and blood supply of the parts.

To correlate his excellent chapter on renal anomalies, the author first enters into a clear description of the development of the kidney. He dwells at length on the anomalous blood supplies of kidneys.

Nitrous oxide gas, with no preliminary sedative, seems to be the favorite anæsthetic; however, much ethylene is also used. Regional anæsthesia is briefly described.

After discussing the instruments used in renal surgery, he writes in detail the indications for and the technique of each method of approach to the kidney. The lumbar extraperitoneal route is usually given first choice. The author recommends nephrotomy for exploratory purposes and concludes that its effect upon the renal parenchyma is a slight loss of renal tissue through scar, but the remainder of the renal cells undergo compensatory hypertrophy.

Posterior and anterior pyelotomy are stressed from the viewpoint of producing hæmorrhage of injured anomalous or supernumerary blood vessels. However, all kidney pelvis work seems to be in agreement with recent American ideas.

In nephrectomies, separate ligation of the renal vessels is preferred. Clamps which are left on the pedicle are used only when nothing else can be used. In secondary nephrectomies the subcapsular method is recommended. Adhesions, large kidneys, hæmorrhage, abnormal vessels, wounds of the inferior vena cava, opening of the pleural cavity, wounds of the diaphragm and nerves, opening of the peritoneum and intestines, and injury to the suprarenals are all pointed out as complications of nephrectomies. Immediate postoperative accidents are operative shock, anuria, hæmorrhage, hæmaturia, and wound infections; late accidents are fistulæ and herniæ of the abdominal wall.

In nephrectomy for tuberculosis the author advocates complete removal of the fatty capsule, cauterization of the ureteral stump, and drainage of the wound.

For nephropexy, the methods of Guyon and Edebohls are mentioned first. The author supplements their methods by using the fatty capsule for a basket to maintain fixation. Plastic operations on the kidney pelvis are fully described but no opinion is given as to the efficacy of the different methods of procedure.

Surgical procedures in nephritis include decapsulation and omental implantation of the kidney. Experimentally, better results are obtained with the latter procedure.

The author concludes that unilateral ligature of a ureter always causes hydronephrosis of the same kidney with a toxic nephritis of the opposite organ. Bilateral ureteral ligature causes death of a uræmic nature. Ligating any renal artery, small or large, causes death of that portion of the kidney tissue supplied by the ligated artery. Anomalous veins or even the renal vein can be ligated with a rather satisfactory formation of collateral venous circulation.

It is stated that in all methods of deviating the urine through paths other than the bladder, that is, leading the ureters to the skin, or transplanting the ureters or entire trigone into the bowel at various sites, always leads to a certain degree of hydronephrosis and a certain amount of pyelonephritis. The illustrations of ureteral transplantation are well worth reviewing.

These two volumes are highly recommended to anybody who has a reading knowledge of French and who is especially interested in surgery of the kidney.

HARRY CULVER.

THE past 15 years have seen a material change in the literature of malignant tumors. Previous to that time much attention was given to various theories of the origin of tumors, the best known of which was Cohnheim's theory of embryonal tissue displacement. All of these theories were largely the result of abstract reasoning based upon histological observations. Today, the study of the etiology of malignant tumors tends to ignore histological material and to be directed along three different lines of investigation: (1) the transplantation of tumors from one animal into another; (2) the relation of heredity to the incidence of tumors; and (3) the experimental production of tumors in lower animals.

Lewin, in this volume[2] of 231 pages, has reviewed the literature on the etiology of malignant tumors. The extent of his labors is indicated by the fact that at the end of the volume is a bibliography of 23 closely printed pages giving references only, and not titles of the articles cited. American medical literature is rather well represented. Carrel, Jorstad, Lambert, Levin, L. Loeb, Murphy, Rous, Slye, Tyzzer, Woglon, and Wells, each has 2 or more citations. The author is convinced that, regardless of any possible microbic origin, the problem of the etiology of malignant tumors stands in close relation to immunobiological processes. In the development of malignant tumors there are two factors, namely: an extrinsic factor (irritation) and an intrinsic factor (inherited disposition, including immunological phenomena).

[1] CHIRURGIE DU REIN. vols. i and ii. By Dr. Edmond Papin. Paris: Gaston Doin et Cie, 1928.

[2] DIE AETIOLOGIE DER BOESARTIGEN GESCHWUELSTE; NACH DEM GEGENWAERTIGEN STANDE DER KLINISCHEN ERFAHRUNG UND DER EXPERIMENTELLEN FORSCHUNG. By Professor Dr. Carl Lewin. Berlin: Julius Springer, 1928.

Lewin's book is divided into five sections. After a short introduction, the author considers at some length (117 pages) the theories of tumor formation and their experimental basis. In this section is considered the experimental evidence that has been educed for and against the older theories of Virchow, Cohnheim, Ribbert, and others. Following this, he discusses tumors induced by mechanical irritants such as diatomaceous earth, light, X-rays, radium, etc. Thirty-six pages are devoted to a consideration of carcinomata induced by chemical irritants, especially tar; 30 pages to transplantation of tumors; and 36 pages to "parasites as causes of malignant tumors."

In the third section of 30 pages are considered the endogenous factors in tumor formation,—constitution, disposition, and natural and acquired immunity. In this section, Miss Slye's work on the relation of heredity to tumors is considered. Here also are considered the relation of the endocrine glands, age, race, tissue and cell disposition, and diet to tumor formation; and finally, the biochemical basis of cancer disposition, and the immunological phenomena of malignant tumors.

The fourth section (25 pages) is devoted to a discussion of the nature of the action of irritants as indicated in the morphological and biochemical changes in cells, and the biology of malignant cells as revealed in their artificial cultivation.

The final division of only 1 page is a statement of the author's conclusion. He does not consider any of the morphological changes in the cells of a malignant tumor as diagnostic of malignancy. It is possible that the biochemical changes in the cells are the real determinants of malignant character. Yet, it is none too certain that these changes may not be effects rather than causes.

This small volume does two things reasonably well: (1) It summarizes the enormous literature on the etiology of malignant tumors in a satisfactory and critical manner. In other words it is a fairly successful attempt at stock-taking. (2) It delimits with considerable accuracy, although without any apparently conscious attempt to do so, the lines of research which may be expected in the future to yield the greatest results. J. P. SIMONDS.

THE second part of the second and concluding volume of the revised second edition of the monumental and epochal work of Czerny and Keller on the feeding of infants and children[1] is before me.

The first edition began to appear in about 1906 and at once established itself as the Bible of child nutrition. The second edition maintains that position; it is the *sine qua non* of the pediatrician's library. As Finkelstein said in his review of the first edition (I am quoting from memory): "Whoever, hereafter, occupies himself with the problems of nutrition in infancy and childhood will find in this work his foundations."

While encyclopedic in scope, it is peculiarly original and stimulatingly unconventional throughout. To those still struggling with a foreign language, its simplicity of diction will have an especial appeal. The position of this volume is as unique and fundamental today as it was twenty years ago.

JOSEPH BRENNEMANN.

IN a short, clear, and well-arranged book,[2] Tylecote and Fletcher—evidently men of wide clinical experience—have dealt with the diagnosis and medical treatment of diseases of the lungs. The book is written for the clinician, emphasis being placed upon clinical signs and symptoms rather than upon micropathology, bacteriology, etc. The book has been written for "the senior student or the young practitioner."

There are no illustrations, charts, or roentgenograms. In this book written in 1927, the authors have availed themselves surprisingly little of the advantages of surgery in the treatment of diseases of the lung. RALPH B. BETTMAN.

IN 1890, long before the value of the collapse treatment of the lung for tuberculosis was generally appreciated, Stuart Tidey[3] conceived the idea of strapping the chest wall with adhesive, for the purpose of aiding the natural processes of healing, by reducing the mobility of the lung and by relieving the affected parts of the natural pull exercised by the chest wall. The beneficial results which he obtained prompted him to bring the matter to the attention of his medical colleagues in an article which was published in the *British Medical Journal* in 1896.

Two years later he met Dr. Forlanini and was invited to visit the great Italian at his clinic, which was "small, having only twenty-five beds," and there saw cases treated with artificial pneumothorax. Later he corresponded with Forlanini and in 1905 received a letter from the originator of the artificial pneumothorax treatment from which the following is quoted: "I see that in America some other physicians such as Lemke and Schnell, following Murphy, have carried out the same treatment, and in Europe all quote these as the originators of it and no one remembers my name, so that the method to which I have prior claim, is called by all 'Murphy's method.'" Lemke holds a different view. . . . But as he thinks it sufficient to obtain rest and not, as I think necessary, absolute immobility, rigorously maintained for a long period, even 2, 3, or 4 years, he limits himself to inducing pneumothorax once or twice in the course of treat-

[1] DES KINDES ERNAEHRUNG, ERNAEHRUNGSSTOERUNGERN UND ERNAEHRUNGSTHERAPIE; EIN HANDBUCH FUER AERZTE. By Professor Ad. Czerny and Professor A. Keller. vol. ii, Part 2. 2d ed., completely rev. Leipzig and Vienna: Franz Deuticke, 1928.

[2] DIAGNOSIS AND TREATMENT IN DISEASES OF THE LUNGS. By Frank E. Tylecote, M.D., D.P.H. (Vict.), F.R.C.P. (Lond.), and George Fletcher, M.A., M.D. (Glas.), M.R.C.P. (Lond.), D.P.H. (Camb.). New York and London: Oxford University Press, 1927.

[3] COMPRESSION OF THE LUNG IN THE TREATMENT OF PULMONARY LESIONS. By Stuart Tidey, M.D. (Lond.), M.R.C.P. (Lond.). New York: Oxford University Press, 1927.

ment, at intervals of several months. And this is the reason that his results are not encouraging."

In this day when the method of treating unilateral phthisis by extrapleural thoracoplasty has been so widely accepted and the method of artificial pneumothorax so well understood, this booklet by Tidey must be regarded more in the light of a delightful bit of historical reading, even though his indications and his rationale for strapping the chest with adhesive are as good and sound as they ever were.

The reviewer is pleased to have had the opportunity of reading this little monograph and is sure that any one interested in the subject of tuberculosis will also enjoy it. RALPH B. BETTMAN.

RIVIERE, in an excellent little book,[1] has described the history, indications, and technique of artificial pneumothorax in the case of pulmonary tuberculosis. A short article dealing with other surgical procedures, such as phrenic exeresis, pneumolysis, and extrapleural thoracoplasty, is appended.

The description of the technique is very good and if the reader would adhere to the methods and precautions laid down, dangers would be minimized to the lowest possible point. The book is in all senses of the word a practical handbook, although the reviewer feels that it could be made even more practical by the inclusion of additional illustrations, roentgenograms, and photographs.

RALPH B. BETTMAN.

[1] THE PNEUMOTHORAX AND SURGICAL TREATMENT OF PULMONARY TUBERCULOSIS. By Clive Riviere, M.D. (Lond.), F.R.C.P. 2d ed. New York and London: Oxford University Press, 1927.

"THE more one sees of practical surgery the more one is impressed by the relative frequency of unforeseen complications and when one carefully thinks out the cause of the troubles it is only to find that most of them could be prevented." This statement in the preface of Watson's book on the *Fundamentals of the Art of Surgery*[2], expresses the underlying sentiment of the volume. The author definitely states in regard to operative skill: "The success of an operation depends on much more than this; it entails first, a careful and thorough examination of the patient; second, an adequate pre-operative preparation; third, a careful anæsthesia and an equally careful selection of the anæsthetic; fourth, the proper organization and equipment of the place for the operation; fifth, due appreciation of the powers of the patient and his ability to stand the strain to be imposed upon him; sixth, adequate facilities for postoperative treatment, and lastly, directions for the after-care of the patient." These are the topics discussed by the author. His presentation is excellent in that he combines definite information with sufficient of the romance of medicine to make easy and delightful reading.

It may be discreet to call attention to an error on page 249. The author states that Bayliss advises the use of a 6 per cent solution of gum acacia in 9 per cent sodium chloride solution to be given intravenously. This, no doubt, is a typographical error and should read 0.9 per cent sodium chloride solution. J. A. WOLFER.

[2] FUNDAMENTALS OF THE ART OF SURGERY. By John H. Watson, M.B.B.S. (Lond.), F.R.C.S. (Eng.). New York: Paul B. Hoeber, 1927.

BOOKS RECEIVED

Books received are acknowledged in this department, and such acknowledgment must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers and as space permits.

POLIOMYELITIS; WITH ESPECIAL REFERENCE TO THE TREATMENT. By W. Russell MacAusland, M.D. Philadelphia: Lea & Febiger, 1927.

CONVALESCENCE; HISTORICAL AND PRACTICAL. By John Bryant, M.D. New York: The Burke Foundation, 1927.

PROCEEDINGS OF THE TWENTY-FIRST ANNUAL CONVENTION OF THE ASSOCIATION OF LIFE INSURANCE PRESIDENTS HELD IN THE HOTEL ASTOR. New York, December 8 and 9, 1927.

MEDICAL RESEARCH COUNCIL; THE TOXAEMIAS OF PREGNANCY; A CLINICAL AND BIOCHEMICAL STUDY. By J. N. Cruickshank, J. Hewitt, and K. L. Couper. London: His Majesty's Stationery Office, 1927.

AIDS TO GYNÆCOLOGY. By Richard E. Tottenham, B.A., M.D., D.P.H.(Univ. of Dublin), F.R.C.P.I. 7th ed. New York: William Wood and Company, 1927.

LE FRATTURE DEL GOMITO. By Dott. Franco Rossi. (Publication of the Clinica Chirurgica Della Regia Universita di Milano.) Milan: Coop. Grafica Degli Opera, 1927.

LAS PERITONITIS POR PERFORACION. By Dr. Elpidio Stincer. Havana: Compania Impresora Industrial, 1927.

BIRTH, STILLBIRTH, AND INFANT MORTALITY STATISTICS FOR THE BIRTH REGISTRATION AREA OF THE UNITED STATES, 1925. Eleventh Annual Report—Part I: Summary and Rate Tables and General Tables. Washington: United States Government Printing Office, 1927.

THE ART OF ANÆSTHESIA. By Paluel J. Flagg, M.D. 4th ed. rev. Philadelphia and London: J. B. Lippincott Company, 1928.

LEHRBUCH DER ROENTGENDIAGNOSTIK MIT BESONDERER BERUECKSICHTIGUNG DER CHIRURGIE. Edited by H. R. Schinz with the collaboration of W. Baensch and E. Friedl. Leipzig: Georg Thieme, 1928.

KURZE GESCHICHTE DER CHIRURGIE. By W. von Brunn. Berlin: Julius Springer, 1928.

TROUBLES WE DON'T TALK ABOUT! By J. F. Montague, M.D., F.A.C.S. Philadelphia, London, Chicago, Montreal: J. B. Lippincott Company, 1927.

GYNÆCOLOGY; FOR STUDENTS AND PRACTITIONERS. By Thomas Watts Eden, M.D., C.M. (Edin.), F.R.C.P. (Lond.), F.R.C.S. (Edin.), and Cuthbert Lockyer, M.D. B.S., F.R.C.S., F.R.C.P. 3d ed. New York: The Macmillan Company, 1928.

CLINICAL RESEARCHES IN ACUTE ABDOMINAL DISEASE. By Zachary Cope, B.A., M.D., M.S. (Lond.), F.R.C.S. (Eng.). 2d ed. London and New York: Oxford University Press, 1927.

BACTERIOLOGY AND SURGERY OF CHRONIC ARTHRITIS AND RHEUMATISM; WITH END-RESULTS OF TREATMENT. By H. Warren Crowe, D.M., B.Ch. (Oxon.), M.R.C.S., L.R.C.P. The Chapter on Surgical Treatment by Herbert Frankling, C.B.E., M.R.C.S. (Eng.). London and New York: Oxford University Press, 1927.

LES TROUBLES FONCTIONNELS DE L'APPAREIL GÉNITAL DE LA FEMME; ÉTUDE PHYSIOLOGIQUE, CLINIQUE, ET THÉRAPEUTIQUE. By Gaston Cotte. Paris: Masson et Cie, 1928.

L'ANNÉE OBSTÉTRICALE; TRAVAUX DE 1925 ET QUESTIONS OBSTÉTRICALES D'ACTUALITÉ. By H. Vignes and B. Jean in collaboration with V. Robin. Paris: Masson et Cie, 1927.

MODERN BAKING POWDER: AN EFFECTIVE, HEALTHFUL LEAVENING AGENT; INCLUDING THE OCCURRENCE OF ALUMINUM COMPOUNDS IN FOODS AND THEIR EFFECT ON HEALTH. Compiled by Juanita E. Darrah, A.B., A.M. Chicago: The Commonwealth Press, Inc., 1927.

PHYSICAL DIAGNOSIS. By Charles Phillips Emerson, A.B., M.D., Philadelphia and London: J. B. Lippincott Company, 1928.

GYNECOLOGY. By Howard A. Kelly, A.B., M.D., LL.D., and collaborators. New York and London: D. Appleton and Company, 1928.

UNIVERSITY OF IOWA STUDIES; STUDIES IN MEDICINE. Vol. iii, No. 1. Iowa City: Published by the University.

CRAWFORD W. LONG AND THE DISCOVERY OF ETHER ANESTHESIA. By Frances Long Taylor. New York: Paul B. Hoeber, 1928.

THE PEAKS OF MEDICAL HISTORY; AN OUTLINE OF THE EVOLUTION OF MEDICINE FOR THE USE OF MEDICAL STUDENTS AND PRACTITIONERS. By Charles L. Dana, A.M., M.D., LL.D. 2d ed. New York: Paul B. Hoeber, 1928.

THE YOUNG MAN AND MEDICINE. By Lewellys F. Barker, M.D., LL.D. New York: The Macmillan Company, 1928.

THE MECHANICS OF THE DIGESTIVE TRACT; AN INTRODUCTION TO GASTROENTEROLOGY. By Walter C. Alvarez, M.D. 2d ed. New York: Paul B. Hoeber, 1928.

LOCAL ANESTHESIA; A SHORT COURSE FOR STUDENTS AND SURGEONS. By Géza de Takáts, M.D., M.S. With a Foreword by Allen B. Kanavel, A.B., M.D., D.Sc. Philadelphia and London: W. B. Saunders Company, 1928.

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